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TRANSACTIONS

OF THE

AMERICAN

SURGICAL ASSOCIATION.

VOLUME THE THIRD.

EDITED BY

J. EWING MEARS, M.D.,

RECORDER OF THE ASSOCIATION.

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OFFICERS

OF THE

AMERICAN SURGICAL ASSOCIATION.

Elected at the meeting, April 24, 1885.

PRESIDENT.

MOSES GUNN, A.M, M.D., LL.D.

VICE-PRESIDENTS.

CHRISTOPHER JOHNSTON, M.D.,
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L. McLANE TIFFANY, A.M. CANTAB., M.D.

LIST OF THE PRESIDENTS

OF THE

ASSOCIATION FROM THE TIME OF ITS INSTITUTION.

ELECTED

1880. SAMUEL DAVID GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., LL.D. Edin.—Founder of the Association

1883. EDWARD MOTT MOORE, M.D., LL.D.

1884. WILLIAM THOMPSON BRIGGS, M.D.

1885. MOSES GUNN, A.M., M.D., LL.D.

FELLOWS

OF THE

AMERICAN SURGICAL ASSOCIATION.

APRIL, 1885.

* Denotes Original Members.
† Denotes Fellows who have resigned.
‡ Denotes Fellows who are deceased.

ELECTED

1882. AGNEW, D. HAYES, M.D., LL.D., 1611 Chestnut Street, Philadelphia. Professor of Surgery and of Clinical Surgery in the University of Pennsylvania, Surgeon to the Pennsylvania Hospital, to the University Hospital, and Consulting Surgeon to the Orthopædic Hospital, Philadelphia.

*1880. ASHHURST, JOHN, Jr., M.D., 2000 West DeLancey Place, Philadelphia. Professor of Clinical Surgery in the University of Pennsylvania, Senior Surgeon to the Children's Hospital of Philadelphia.

1882. BARTON, JAMES M., A.M., M.D., 1344 Spruce Street, Philadelphia. Surgeon to the Jefferson College Hospital, and to the German Hospital, Philadelphia.

*1880. BENHAM, SILAS NELSON, A.M., M.D., 156 Third Avenue, Pittsburgh, Pa.

1882. BILLINGS, JOHN SHAW, M.D., LL.D. Edin., Surgeon-General's Office, Washington. Surgeon U. S. Army. *Member of Council*, 1884–85.

1882. BONTECOU, REED BROCKWAY, M.D., 68 Fourth Street, cor. Broadway, Troy, N. Y. Surgeon to Marshall Infirmary of Troy.

1882. BOZEMAN, NATHAN, M.D., 296 Fifth Avenue, New York. Surgeon to the Woman's Hospital of the State of New York.

ELECTED

*1880. Bramble, D. D., M.D., 169 Broadway, Cincinnati, Ohio.

*1880. Briggs, William Thompson, M.D., corner Summer and Union Streets, Nashville, Tenn. Professor of Surgery in the Medical Department of the University of Nashville and Vanderbilt University, Surgeon to the University Hospital of Nashville. *President*, 1884.

*1880. Brinton, John H., M.D., 1423 Spruce Street, Philadelphia. Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College of Philadelphia, Surgeon to St. Joseph's Hospital of Philadelphia. *Treasurer*, 1884–85.

*1880. Byrd, William Andrew, M.D., 407 Jersey Street, Quincy, Ill. Professor of Clinical Surgery in the Quincy College of Medicine, Consulting Surgeon to the Sarah Denman Hospital, Quincy, Ill.

1882. Bull, William Tillinghast, M.D., 35 West Thirty-fifth Street, New York. Surgeon to the New York Hospital, to Chambers Street Hospital, Consulting Surgeon to St. Luke's Hospital and to the Hospital for Ruptured and Crippled, New York.

*1880. Cabell, James L., A.M., M.D., LL.D., University of Virginia, Virginia. Professor of Surgery in the University of Virginia.

*1880. Campbell, Henry Fraser, A.M., M.D., Augusta, Ga. Professor of Surgery and Gynæcology in the Medical Department of the University of Georgia, Surgeon to the City Hospital and to the Freedman's Hospital of Augusta, Ga. *Member of Council*, 1882.

1885. Carmalt, William H., A.M., M.D., 87 Elm Street, New Haven, Connecticut. Professor of Surgery in Yale College, Surgeon to the New Haven Hospital.

1882. Cheever, David Williams, M.D., 239 Boylston Street, Boston. Professor of Surgery, Harvard University, Surgeon to the Boston City Hospital.

*1880. Coleman, John Scott, M.D., 563 Green Street, Augusta, Georgia.

1881. Cole, Richard Beverly, M.D., Ph.D., San Francisco, Cal.

ELECTED

1882. COMINGOR, JOHN A., M.D., 77½ South Illinois Street, Indianapolis, Ind. Professor of Surgery in the Indiana Medical College, Surgeon to the City Hospital of Indianapolis.

*1880. CONNER, PHINEAS SANBORN, M.D., LL.D., 159 West Ninth Street, Cincinnati, Ohio. Professor of Anatomy and of Clinical Surgery in the Medical College of Ohio, Professor of Surgery in the Medical Department of Dartmouth College, Hanover, N. H., Surgeon to the Cincinnati Hospital and to the Good Samaritan Hospital, Cincinnati. *Member of Council*, 1883.

1882. CUNNINGHAM, FRANCIS DEAN, M.D., 717 Franklin Street, Richmond, Va.

1883. DANDRIDGE, NATHANIEL PENDLETON, M.D., No. 2, Oritz, Fourth and Sycamore Streets, Cincinnati, Ohio. Professor of Genito-Urinary Diseases in Miami Medical College, Surgeon to the Cincinnati Hospital and to the Episcopal Hospital for Children, Cincinnati.

1882. DAVIS, J. S., M.D., University of Virginia, Virginia.

*1880. DAVIS, SAMUEL T., M.D., 134 North Fourth Street, Lancaster, Pa.

*1880. DAWSON, WILLIAM WIRT, M.D., corner Third and Broadway, Cincinnati, Ohio. Professor of Surgery in the Medical College of Ohio, Surgeon to the Hospital of the Good Samaritan, Cincinnati. *Vice-President*, 1883.

1882. DENNIS, FREDERICK SHEPARD, M.D., 21 East Twenty-first Street, New York. Professor of the Principles and Practice of Surgery in the Bellevue Hospital Medical College, Surgeon to the Bellevue Hospital, to St. Vincent's Hospital, and Consulting Surgeon to the Hospital for Ruptured and Crippled, New York.

1882. DETMOLD, WILLIAM, M.D., 38 East Ninth Street, New York. Professor of Military and Clinical Surgery in the College of Physicians and Surgeons of Columbia College (Emeritus), Consulting Surgeon to the Presbyterian Hospital of New York.

1882. DUNOTT, THOMAS J., M.D., 132 Walnut Street, Harrisburg, Pa. Surgeon to the Harrisburg Hospital.

ELECTED

1883. Fenger, Christian, M.D., 214 East Ohio Street, Chicago. Professor of General Pathology and Pathological Anatomy in the Chicago Medical College (Medical Department of Northwestern University).

1882. Fifield, William Cranch Bond, M.D., M.R.C.S. Lond., Harrison Square, Boston. Surgeon to the Boston City Hospital.

1882. Forbes, William Smith, M.D., 1409 Locust Street, Philadelphia. Demonstrator of Anatomy in the Jefferson Medical College of Philadelphia, Surgeon to the Protestant Episcopal Hospital of Philadelphia.

1882. Gay, George Washington, M.D., 589 Tremont Street, Boston. Surgeon to the Boston City Hospital. *Member of Council*, 1882.

*1880. Gouley, John W. S., M.D., 134 Madison Avenue, New York. Surgeon to Bellevue Hospital, New York. *Member of Council*, 1881.

1882. Gregory, Elisha H., M.D., LL.D., 1006 Olive Street, St. Louis. Professor of Surgery in the St. Louis Medical College, Surgeon to the St. Louis Hospital, St. Louis. *Vice-President*, 1884.

*1880. Gross, Samuel W., A.M., M.D., 1112 Walnut Street, Philadelphia. Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College of Philadelphia.

*1880. Gunn, Moses, A.M., M.D., LL.D., 2101 Calumet Avenue, Chicago. Professor of Surgery in Rush Medical College, Surgeon to St. Joseph's Hospital, Chicago. *Member of Council*, 1880; *Vice-President*, 1882; *President*, 1885.

1882. Hewson, Addinell, M.D., 301 South Fifteenth Street, Philadelphia.

*1880. Hutchison, Joseph Chrisman, M.D., LL.D., 130 Hicks Street, Brooklyn, N. Y. Surgeon to the Brooklyn City Hospital and to the Brooklyn Orthopædic Infirmary, Consulting Surgeon to the Kings County Hospital, to St. John's Hospital, and to St. Peter's Hospital, Brooklyn. *Member of Council*, 1880–81; *Vice-President*, 1884.

ELECTED

1882. Isham, Ralph N., A.M., M.D., 321 Dearborn Avenue, Chicago. Professor of the Principles and Practice of Surgery in the Chicago Medical College (Medical Department of the Northwestern University).

*1880. Johnston, Christopher, M.D., Baltimore. Professor of Surgery in the University of Maryland (Emeritus). *Vice-President*, 1885.

*1880. Keller, James McDonald, M.D., Hot Springs, Arkansas.

*1880. Keen, William Williams, A.M., M.D., Philadelphia. Professor of the Principles and Practice of Surgery in the Woman's Medical College of Philadelphia, Surgeon to St. Mary's Hospital of Philadelphia.

*1880. Kinloch, Robert Alexander, M.D., 209 Meeting Street, Charleston. Professor of Surgery in the Medical College of the State of South Carolina, Surgeon to the Roper Hospital, to the City Hospital, and Surgeon and Gynæcologist to St. Xavier's Infirmary, Charleston. *Member of Council*, 1880–81.

*1880. Levis, Richard J., A.M., M.D., Northwest corner Walnut and Sixteenth Streets, Philadelphia. Surgeon to the Pennsylvania Hospital and to the Jefferson College Hospital, Philadelphia.

*1880. Marks, Solon, M.D., Milwaukee, Wisconsin. Surgeon to St. Mary's Hospital of Milwaukee.

*1880. Mastin, Claudius Henry, M.D., LL.D. Univ. Penn., 110 St. Francis Street, Mobile, Ala. Surgeon to the Providence Infirmary, Mobile, Alabama. *Vice-President*, 1883.

1883. McCann, James, M.D., 300 Penn Avenue, Pittsburgh. Surgeon to the Western Pennsylvania Hospital, Pittsburgh, Pennsylvania.

1882. McGraw, Theodore A., M.D., 107 West Fort Street, Detroit, Michigan. Professor of Surgery in the Detroit Medical College, Surgeon to St. Mary's Hospital and to the Harper Hospital, Detroit.

ELECTED

*1880. McGuire, Hunter, M.D., 513 Grace Street, Richmond, Va. *Member of Council*, 1880–85.

1882. Maclean, Donald, M.D., F.R.C.S. Edin., 72 Layfayette Avenue, Detroit, Michigan. Fellow of the Royal College of Physicians and Surgeons, Kingston, Ontario, Professor of Surgery and of Clinical Surgery in the University of Michigan, Surgeon to the University Hospital, Ann Arbor, Consulting Surgeon to Harper Hospital, Detroit.

1882. McLean, LeRoy, M.D., 21 First Street, Troy, N. Y. Surgeon to the Troy Hospital in charge of the Sisters of Charity.

*1880. Mears, J. Ewing, A.M., M.D., 1429 Walnut Street, Philadelphia. Professor of Anatomy and of Clinical Surgery in the Pennsylvania College of Dental Surgery, Demonstrator of Surgery in the Jefferson Medical College of Philadelphia, Surgeon to St. Mary's Hospital, and Gynæcologist to the Jefferson Medical College Hospital of Philadelphia. *Recorder*, 1881–85.

1885. Michael, Jacob Edwin, A.M., M.D., 246 Madison Avenue, Baltimore. Professor of Anatomy and Clinical Surgery in the University of Maryland, Surgeon to the University Hospital, Consulting Surgeon to the Presbyterian Eye, Ear, and Throat Charity Hospital, Baltimore.

*1880. Moore, Edward Mott, M.D., LL.D., 63 South Fitzhugh Street, Rochester, N. Y. Surgeon to St. Mary's Hospital of Rochester. *Vice-President*, 1882; *President*, 1884.

*1880. Morton, Thomas George, M.D., Philadelphia. Professor of Clinical and of Operative Surgery in the Philadelphia Polyclinic and College for Graduates, Surgeon to the Pennsylvania Hospital, to the Orthopædic Hospital, and to the Jewish Hospital, Philadelphia.

1882. Nancrede, Charles Beylard, M.D., 2109 Pine Street, Philadelphia. Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic and College for Graduates, Surgeon to the Protestant Episcopal Hospital and to St. Christopher's Hospital for Children, Philadelphia.

1882. Norris, Basil, M.D., Medical Director U. S. Army, San Francisco, California.

ELECTED

1882. Owens, John E., M.D., 1806 Michigan Avenue, Chicago. Professor of Surgical Anatomy and Operative Surgery in the Chicago Medical College, Attending Surgeon to St. Luke's Hospital of Chicago.

*1880. Packard, John H., M.D., 1926 Spruce Street, Philadelphia. Surgeon to the Pennsylvania Hospital and to St. Joseph's Hospital, Philadelphia. *Treasurer*, 1880–83.

*1880. Pancoast, William Henry, M.D., 1500 Walnut Street, Philadelphia. Professor of Anatomy in the Jefferson Medical College of Philadelphia.

1885. Park, Roswell, A.M., M.D., 305 Delaware Street, Buffalo, New York. Professor of Surgery in the Medical Department of the University of Buffalo, Surgeon to the Buffalo General Hospital.

1882. Parkes, Charles T., M.D., 125 State Street, Chicago. Professor of Anatomy in Rush Medical College, Surgeon to the Presbyterian Hospital, Physician-in-Charge of St. Joseph's Hospital, Chicago.

1883. Peck, Washington F., A.M., M.D., Davenport, Iowa. Professor of Surgery and of Clinical Surgery in the Medical Department of the State University of Iowa, Surgeon to the Mercy Hospital of Davenport, and Consulting Surgeon to the Mercy Hospital of Iowa City.

1881. Pollock, Alex. M., M.D., Pittsburgh, Pa. Surgeon to the Mercy Hospital and to the Passavant's Hospital of Pittsburgh.

1882. Porter, William Gibbs, M.D., 1223 Spruce Street, Philadelphia. Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1882. Prewitt, Theodore F., M.D., 2126 Olive Street, St. Louis. Professor of the Principles and Practice of Surgery and of Clinical Surgery in the Missouri Medical College, Surgeon to St. John's Hospital, St. Louis.

1882. Prince, David, M.D., Jacksonville, Illinois.

1882. Reed, Thomas B., M.D., 1427 Walnut Street, Philadelphia. Surgeon to the Presbyterian Hospital, Philadelphia.

ELECTED

1882. Roberts, John B., M.D., 1118 Arch Street, Philadelphia. Professor of Applied Anatomy and of Operative Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine, Surgeon to St. Mary's Hospital of Philadelphia.

*1880. Richardson, Tobias G., M.D., New Orleans. Professor of Surgery in the Medical Department of the University of Louisiana, Surgeon to the Charity Hospital, New Orleans.

1882. Rushmore, John Dikeman, M.D., 129 Montague Street, Brooklyn, N. Y. Professor of Surgery in the Long Island College Hospital of Brooklyn, Surgeon to the Brooklyn Hospital, to St. Peter's Hospital, and to the Brooklyn Eye and Ear Hospital.

*1880. Russell, Thomas Pember, M.D., Oshkosh, Wisconsin. *Vice-President*, 1885.

1882. Sabine, Thomas Taunton, M.D., 8 East Twenty-fourth Street, New York. Professor of Anatomy in the College of Physicians and Surgeons, New York.

*1880. Sayre, Lewis Albert, M.D., 285 Fifth Avenue, New York. Professor of Orthopædic Surgery in the Bellevue Hospital Medical College, Consulting Surgeon to the Bellevue Hospital, to the Charity Hospital, to St. Elizabeth's Hospital, and to the Home for Incurables, New York.

1882. Senn, Nicholas, M.D., Milwaukee, Wis. Surgeon to the Milwaukee Hospital.

1882. Smith, Alan Penniman, M.D., 45 Franklin Street, Baltimore, Md. Surgeon to St. Vincent's Hospital, to the Union Protestant Infirmary, to the Hebrew Hospital, to St. Agnes's Hospital, to the Maryland Eye, Ear, and Throat Hospital, and to the Hospital for Diseases of Women, Baltimore, Maryland.

1882. Smith, Stephen, A.M., M.D., 31 West Forty-second Street, New York. Professor of Clinical Surgery in the University Medical College of New York, Surgeon to Bellevue Hospital and to St. Vincent's Hospital, New York.

1882. Squire, T. H., M.D., 409 East Church Street, Elmira, N. Y.

*1880. Swinburne, John, M.D., 57 Eagle Street, Albany, N. Y.

ELECTED

1883. Taylor, William Edwin, M.D., "The Baldwin," San Francisco, Cal. Professor of the Principles and Practice of Surgery in the Medical Department of the University of California, and in the Dental Department of the same University, San Francisco.

1882. Thomson, William, M.D., 1502 Locust Street, Philadelphia. Honorary Professor of Ophthalmology in the Jefferson Medical College of Philadelphia, Ophthalmic Surgeon to the Jefferson Medical College Hospital, Philadelphia.

1885. Thompson, J. Ford, M.D., 1401 H Street, N. W., Washington, D. C., Professor of Surgery in the National Medical College, Surgeon to the Garfield Memorial Hospital and the Children's Hospital.

1882. Thompson, J. W., M.D., Paducah, Kentucky.

1882. Tiffany, Louis McLane, A.M. Cantab., M.D., 137 Park Avenue, Baltimore. Professor of Surgery in the University of Maryland, Surgeon to the University Hospital, Baltimore. *Member of Council*, 1885.

*1880. Tremaine, William S., M.D., Buffalo, N. Y. Major and Surgeon U. S. Army. Professor of Surgery in the Niagara University of Buffalo, N. Y., Surgeon to the Buffalo Hospital of Sisters of Charity, Buffalo, N. Y.

1882. Vanderveer, Albert, A.M., M.D., Ph.D., 28 Eagle Street, Albany, N. Y. Professor of Surgery and Clinical Surgery in the Albany Medical College, Consulting Surgeon to St Peter's Hospital, and Attending Surgeon to the Albany Hospital.

1885. Varick, Theodore R., M.D., 239 Washington Street. Jersey City, N. J. Surgeon to St. Francis's Hospital and to Jersey City Charity Hospital.

*1880. Wales, Philip Skinner, M.D., Surgeon U. S. Navy, 813 Vermont Avenue, Washington, D. C.

1882. Warren, John Collins, M.D., 58 Beacon Street, Boston. Assistant Professor of Surgery in Harvard University, Surgeon to Massachusetts General Hospital, Boston.

ELECTED

1882. Watson, B. A., A.M., M.D., 124 York Street, Jersey City, N. J. Surgeon to the Jersey City Charity Hospital, to St. Francis's Hospital, and to Christ's Hospital.

*1880. Weist, Jacob Rowland, A.M., M.D., 118 North Eighth Street, Richmond, Ind. Surgeon to St. Stephen's Hospital, Richmond, Ind. *Secretary*, 1880–85.

*1880. Westmoreland, W. F., M.D., Atlanta, Ga.

1882. White, J. William, M.D., Ph.D., 222 South Sixteenth Street, Philadelphia. Demonstrator of Surgery, Lecturer on Operative and Minor Surgery, Lecturer on Genito-Urinary and Venereal Diseases in the University of Pennsylvania, Surgeon to the Philadelphia Hospital, and Assistant Surgeon to the University Hospital, Philadelphia.

1882. Willard, DeForest, M.D., Ph.D., 1818 Chestnut Street, Philadelphia. Lecturer on Orthopædic Surgery in the University of Pennsylvania, Surgeon to the Presbyterian Hospital, and Consulting Surgeon to the Home for Cripples, Philadelphia.

*1880. Yandell, David W., M.D., Louisville, Ky. Professor of the Principles of Surgery and of Clinical Surgery in the Medical Department of the University of Louisville.

HONORARY FELLOWS.

1885. Annandale, Thomas, Regius Professor of Clinical Surgery in the University of Edinburgh.

‡1882. Atlee, John L., M.D., LL.D., Lancaster, Pa.

1885. Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna.

1885. Czerny, Vincenz, M.D., Professor of Surgery in the University of Heidelberg.

1885. Erichsen, John Eric, LL.D., Emeritus Professor of Surgery in the University College, London.

ELECTED

1885. Esmarch, Friedrich, M.D., Professor of Surgery in the University of Kiel.

1882. Horwitz, P. J., M.D., Medical Director U. S. Navy, Philadelphia.

1882. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital, Philadelphia.

1885. Lister, Sir Joseph, Bart., D.C.L., LL.D., Professor of Clinical Surgery in King's College, London.

1885. Nussbaum, Dr. V., Professor of Surgery in Munich.

1885. Ollier, Léopold, M.D., Professor of Clinical Surgery at the Faculty of Medicine of Lyons.

1885. Paget, Sir James, Bart., D.C.L., LL.D., London.

1885. Verneuil, A., M.D., Professor of Clinical Surgery at the Faculty of Medicine of Paris.

1885. Volkmann, Richard, M.D., Professor of Surgery at the University of Halle.

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NOTICE.

This volume of Transactions contains the papers read before the Association at the meeting held April 21 to 24, 1885.

The Association assumes no responsibility for the statements and opinions published in this volume.

ANNUAL ADDRESS

BY THE PRESIDENT,
WILLIAM T. BRIGGS, M.D.

[Delivered at the meeting held in Washington, D. C., April 21, 1885.]

Fellows of the American Surgical Association:—

In assuming the duties of the Presidency of this body, I beg to repeat the expression of my profound appreciation of the exalted honor which your kind partiality elicited at the time of my election a year ago. It is, if possible to be so, more sensibly felt now, as I stand to-day in this representative assembly of American Surgeons, than then.

The intervening period has been passed in our respective fields of labor, and doubtless has been employed in the acquisition of further contributions which you are prepared to lay on this common altar erected for that purpose. I greet you officially, as we have met each other socially in the renewal of personal friendships, with the cordial hand-grasp of our professional fellowship, and felicitate you on the promise of a successful session.

We may safely indulge the confident expectation that this meeting will be characterized by the spirit in which the Association had its origin, and will be pervaded by the same united purpose, the same harmonious and agreeable intercourse and similar valuable work to that which has edified and instructed us in previous meetings, and which, in the brief period of its existence, has so illustrated the usefulness of the Association, that its permanency as an exponent of the status of American Surgery, and as a factor in the true advancement of its science and art is fully assured.

Notwithstanding these just and bright anticipations on the threshold of this session, we feel oppressed and saddened by the notable absence of a Fellow who, when among us, stood like

"Saul, a head and shoulders taller than his brethren," an absence which deprives us of the light of remarkable genius and learning upon our discussions, and at the same time bows our heads in sorrow for the loss of one whom we had come to venerate as the Nestor of American Surgery.

While we sat a year ago, his noble form lay prostrate and his spirit even then was passing, though we had hoped that he might be spared. To-day we realize that he whose fertile brain conceived, and whose indomitable effort brought this Association into existence and developed it to its present proud position among the scientific bodies of the country, has gone from among us forever. The death of Prof. Samuel David Gross orphaned the surgical profession of America, and removed from its ranks one who, as pathologist, surgeon, and medical philosopher, was the acknowledged peer of any one in the world. He no longer lives in the flesh, but his labors in the domain of science for a period of nearly sixty years have secured an immortality of fame, and his utterances and achievements will descend to future generations of the profession as a precious heritage. It may be justly said of him, not only that he was the greatest of American surgeons, for that proud eminence is conceded by all, but that he was a *great man.* The deference and honor paid to him the world over, and the specific distinction he bore as the gift of the two most ancient and renowned institutions of learning, teaching in the English tongue, attest the exalted reputation as a scholar and scientist he had won. But he had titles to greatness other than these, and in a broad and philosophic sense superior to them. Though endowed with brilliant intellectual qualities, his life and example are truly great in that he conspicuously illustrated the value and dignity of enthusiastic labor and devotion to his profession. His publications—not one of them inferior and some of them unequalled —constitute a pyramid of industry.

He was great in displaying the possibilities of perseverance and untiring toil animated by lofty aims, and stands forth an exemplar and majestic model on which aspiring youth may fashion itself to the noblest form of achievement. Before his day

as a surgeon, Physick in the East and Dudley in the West had shone resplendent and without rivalry. He came to fill, and filled in the professor's chair and at the operating table, the respective theatres of both those justly eminent men. Both East and West, New York and Philadelphia, and Louisville and Cincinnati are proud to claim that they were the seats from which he taught and the spheres in which he wrought. In the fulness of his stature it may be said of Gross, that "he whose fame filled the world stood like the statue of a demigod poised on the apex of his monumental shaft, far above all surrounding things, pointing to an earlier day-star than greets the vision of ordinary mortality." He was indeed the Father of this Association. It is the child of his ripe old age, and was to him a deeply cherished offspring. He has left it to us, his Fellows and successors, to nurture and maintain in all the vigor and power with which he would have invested it. Let us then be animated by the same lofty spirit which moved him in its organization, and vow to each other, as in the temple, that we will devote ourselves to the duty of making it as great and shining as the fame of its founder, and as enduring as the sciences to which he consecrated his life.

In the brief address which I have purposed to make in the inauguration of this—the sixth annual session of this Association—a few suggestions have occurred to me worthy of practical consideration, and I trust that in no sense will any of them be held as criticisms on any previous action or method of the Association.

We style our body The American Surgical Association, and claim and aspire to a representative national character. In order, however, that this feature may be attained and acknowledged, is it not proper—nay essential—that every reputable surgeon in our extended country who fulfils the requirements of the constitution should be embraced in the fold of our fellowship? It seems wise to me, that year after year, talented, industrious, and aspiring surgeons should be admitted to our list of members.

The elections should not be indiscriminate by any means, and

the provisions of our organization prohibit that possibility. But it is contended that when a worthy and promising worker in the field of surgery has had judgment passed on him, he should be made a Fellow. We should avoid the character of a close corporation, as such we should deprive the institution of the sympathy and coöperation of the great body of men who are laboring in our special sphere. The annual introduction of new members of the class our constitution contemplates, would tend to add new vigor and force, and deepen and widen the foundation on which we purpose to build. It is a recorded fact, that on several occasions surgeons regarded as the peers of any of us have been ostracized by the ballot.

Let us be candid with ourselves, and ask if it is right or just that we who by accident were founders should arrogate superiority in the specialty and assume to be its exclusive representatives. It is my sincere conviction that the prosperity and usefulness of the Association demand that every vacant Fellowship should be filled with good and true men, as fast as they may be presented, and that no savor of favor, affection, or partiality or tinge of invidiousness, should be justly ascribed to the balloting for membership. Nay, I will go further and say that if the limit of the constitution should be found insufficient to embrace all the worthy and industrious surgeons of the land, its terms should be enlarged to include them. It is no sufficient answer to this appeal to say that the body is yet young and vigorous—strength can only be maintained by the infusion of new blood, and by the addition of new material, and the Association should cordially and promptly appropriate and utilize all the proper available force which the surgical profession of the country may offer.

To the consummation of the work of the Association, it is of the first importance to acquire and constantly recruit a body of Fellows who fully appreciate its worth and dignity, and who may be relied on to apply themselves with diligence, in order that each annual meeting may be furnished with a series of valuable contributions. Every honorable and meritorious surgeon who may be admitted to Fellowship should be inspired to

attend every meeting, and feel that it is incumbent on him to be prepared to discuss every essay presented, and to contribute in every possible mode to the interest and instruction to be derived from our proceedings.

The President's address should be required to be scientific in its character; the discussion of some special topic to be chosen by himself, or designated by resolution of the body, or a *résumé* of the progress of surgery for the previous years.

The Association should take care to guard itself from degenerating into a mere social and convivial gathering. It is pleasant and in the highest degree gratifying to meet each other in the freedom and ease of social fraternity, but this should be made subordinate and incidental to our professional objects, and high above all other sentiments and observances the torch of science should be uplifted in our meetings.

For several years past our annual meetings have been held in April or May. It was the original design, as I conceived, that they should be held at a later period in the year, during what is known as the heated term; and it is suggested that the attendance of a larger number of Fellows, as well as their individual comfort and convenience, would be promoted by the resumption of that arrangement. At the former season we are engaged in active practice, and it is not always possible for Fellows to interrupt their engagements and duties. In August or early in September the members feel the need of a period of relaxation and rest, and usually could so order their work as to enable them to avail themselves of that time, while the meetings could be appointed for some agreeable seaside resort or inland watering place, where rest and enjoyment could be combined with the labor in which we delight—the communion and attrition of mind with mind on scientific subjects. I suggest such a change in the time of meeting to the serious consideration of our Business Committee.

The delay in the appearance of the second volume of the TRANSACTIONS of the Association has been the occasion of regret, and the expression of impatience in some quarters. I think I can assure the Fellows this tardiness was not due to the indifference

or neglect of our very efficient Committee on Publication, and it is surmised that it is to be ascribed to the authors of some of the valuable papers which compose the body of that work. In the larger number of instances, these papers were held for revision and completion, and the committee has been dependent for their prompt return. This difficulty can be obviated and future complaints prevented, if authors of papers will prepare their manuscripts for the press before they are read to the Association, so that they could be at once delivered to the committee. The stenographic notes of discussion should also be promptly written out and submitted for revision. A few weeks, or months at most, would then be sufficient for the appearance and delivery of each volume of the TRANSACTIONS. This is a matter of no little moment, as it is a very natural desire for each Fellow to be enabled to review in print the work of each meeting, before he commences to prepare himself for another. It is to be hoped this suggestion will be acceded to by all, and it may be proper to formulate it as an order of the Association.

The constitution of the Association provides for the election of Honorary Fellows—surgeons of age and distinction, home and foreign, who may or may not have retired from the field of active labor. Thus far in our history, no foreign name has been placed on that list. With five full and prosperous meetings behind us, our body may claim to have passed the stage of adolescence, and assumed the proportions of mature and permanent manhood among the scientific bodies of the world, and it is suggested the Council might very properly consider a few of the most eminent names in foreign countries, on whom this distinction might be conferred. This honor should not be cheapened by lavish and promiscuous distribution, but the recognition of distinguished ability is a highly proper courtesy, and such alliances act and react beneficially to the advancement of science, and promote the sympathy of brotherhood and aspiration among its devotees.

In this beautiful capital city of this great and rapidly growing nation—in science, art, literature, and government, the hundred years' wonder and admiration of older countries and peoples—

you have assembled from all sections of its widely extended territory. You are the distinguished representatives of one of the great scientific professions, and of a special department thereof, whose cultivation in America has already greatly enriched the world's stock of knowledge and skill. You are here to mingle in discussion on its important themes and problems, to beat its accumulated observations with the flail of discernment and truth, and glean the wheat from the chaff, and to vindicate science, by demonstrating for it the justice of the old Roman definition, that it is true knowledge deduced from true principles—*certa cognitio ex principiis certis deducta.* I invoke you to realize the beneficence and nobility of the mission. In such commingling we should strive to elevate and embellish the dignity and value of our specialty as an instrumentality for the alleviation of human suffering by the ripest thought and experience we can bring to its work; to cement the ties of personal friendship which an object so laudable has interwoven; and to add to the renown of this Association.

I need not repeat my congratulations on the success of our institution thus far proven. Its two volumes of TRANSACTIONS assert a high place for it among kindred organizations. They reflect great credit on the Association and demand recognition of the valuable labors of the Committee on Publication. The success of our annual meetings we all feel is greatly due to the able and accomplished Secretary of the Association who has so faithfully performed his task.

In conclusion, Fellows, I again thank you for the high honor you have conferred on me, and bespeaking your generous indulgence for any errors I may commit while occupying the chair, I now declare the Association organized for its session.

REMARKS OF THE PRESIDENT, DR. BRIGGS, ON HIS RETIREMENT FROM OFFICE.

The hour has arrived, gentlemen, for terminating the work of this session, and I may be permitted to congratulate you on the success of this our sixth session. We have had many important papers, and the discussions have been very interesting and instructive. Some of the papers have given evidence of great original research. Our meeting has been harmonious and pleasant, old friendships have been renewed, new ones formed, and we have met in the banqueting hall.

Before dissolving my official connection, I desire to express my great gratification at the selection of my successor, who is not only one of the oldest, but one of the most distinguished of our Fellows.

I desire also to express my pleasure at the re-election of the two officers who have been so efficient in conducting the work of the session to a successful close.

Thanking you for your kindness, gentlemen, I declare this meeting adjourned.

REMARKS BY DR. MOSES GUNN ON HIS ELECTION AS PRESIDENT.

To say that I am deeply sensible and profoundly thankful for the honor conferred upon me is perhaps common-place, and yet certain sentiments are better expressed in the common-place than in any other way. Simplicity if borne out is very harmonious with sincerity. Whatever any one has to say on an occasion like this should be very simple and common-place. There-

fore when I tell you that I am profoundly thankful, I hope that you will accept it as sincere.

When I think of our venerable Fellow, and for three years our President, upon whom had been heaped so many honors, and upon whom they sat so gracefully; when I think that he said that he was proud to be the President of the American Surgical Association; when I think that he said that, and said it in that common-place and simple way, I think that one who is as unworthy as I am, can certainly be pardoned in expressing the same pride that he did.

I therefore thank you for your kindness in placing me in the chair. I accept the trust and promise you that I shall give to it the best efforts that I possess, and that whatever is lacking in any way, will come from want of ability and not from a want of earnest disposition to serve you.

REPORT OF THE COMMITTEE ON NECROLOGY.

SAMUEL DAVID GROSS, M.D., D.C.L. Oxon., LL.D. Cantab., Edin., Jeff., Univ. of Penna.

Emeritus Professor of Surgery in the Jefferson Medical College.
Born July 8, 1805; Died May 6, 1884.

Born at Easton, Pa., July 8, 1805, the Founder of the American Surgical Association received the degree of M.D. from Jefferson Medical College in 1828, and at once began that active life of professional study, practice, writing, and teaching, which, continuing for more than fifty years, closed only with the last weeks of his life.

A profound student, a philosophic investigator, a painstaking experimenter, a careful observer, an accurate diagnostician, a superior operator, a wise counsellor—it was as a teacher and a writer that Professor Gross mightily influenced the surgery of our age.

Demonstrator of Anatomy in the Medical College of Ohio in 1833, he passed in 1835 to the chair of Pathological Anatomy in the Cincinnati College, and in 1840 to that of surgery in the University of Louisville, in which position he remained until his acceptance (in 1856) of the chair of surgery in Jefferson Medical College, with the exception of the winter of 1850–1, when he occupied the surgical chair in the University of New York.

Of commanding presence, with a full deep voice, master of his subject, thoroughly in earnest, methodical in arrangement, simple yet forcible in expression, never for an instant at a loss for a thought or the word—he was an ideal lecturer; and no other American surgeon has ever so impressed himself upon medical students and upon so many.

Translating in the first year of his professional life four medi-

cal works,[1] a year later he was the author of a "Treatise on the Anatomy, Physiology, and Diseases of the Bones and Joints." In 1839 his "Elements of Pathological Anatomy" appeared; in 1843 his "Experimental and Critical Inquiry into the Nature and Treatment of Wounds of the Intestines;" in 1851 his "Practical Treatise on the Diseases, Injuries, and Malformations of the Urinary Bladder, the Prostate Gland, and the Urethra;" in 1854 his "Practical Treatise on Foreign Bodies in the Air-Passages;" and in 1859 (the preface dated on his fifty-fifth birthday) his master work, the "System of Surgery." Though the changes in theory and practice consequent upon increased knowledge and scientific progress have rendered much of the earlier writings of but limited value to the medical man of to-day, yet their evidences of thorough preparation, their methodical arrangement, and their clearness of expression will always secure for them the attention of scholars. The "System of Surgery," almost encyclopædic in character, and bearing such strong marks of its author's learning, wisdom, and sound judgment, will, when it ceases to be the text- and reference-book of our students and practitioners, take its place as one of the very few medical classics of the nineteenth century.

In addition to the volumes named, a great number of published reports, lectures, monographs, addresses, criticisms, editorials, writings upon most varied subjects, prepared for different occasions or inspired by the peculiar demands of the times—testify yet further to the range of study, the depth of thought, and the untiring industry of Professor Gross. No man's life could more clearly prove that in his inmost soul he felt the truth of Velpeau's declaration, "we must ever keep working, my friends." From his very death-bed he sent to our Association a paper on "Wounds of the Intestines," and to the American Medical Association one on "Lacerations of the Female Sexual Organs," each of which bore witness to the fact that though the hand might tremble and the eye be dim, the mind was as clear and active as ever, and the love of professional work undiminished.

[1] Bayle and Hollard's General Anatomy, Hildenbrand on Typhus Fever, Hatin's Manual of Obstetrics, Tavernier's Operative Surgery.

On the 6th of May, 1884, having almost reached the "extreme limits of the Psalmist," full of honors and well-beloved, he rested from his labors, and his works do follow him.

RESOLUTIONS WITH RESPECT TO THE DECEASE OF PROFESSOR GROSS.

WHEREAS, After a long and most distinguished career, Professor Samuel David Gross has passed from earth;

Resolved, That we, the Fellows of the American Surgical Association, of which he was the Founder and First President, recognizing his great learning, his remarkable industry, his surgical pre-eminence, his nobility of character, and his purity of life, give official expression to our profound respect for his memory, and our deep sense of the great loss we have sustained in the removal from among us of a guide, a counsellor, and a most devoted fellow-worker.

Resolved, That as our heartfelt sympathy is with those to whom he was a wise and loving father, a copy of these resolutions be sent to them through his son, our Fellow, Professor Samuel W. Gross.

Resolved, That these resolutions, together with a memorial notice, be published in the next volume of the TRANSACTIONS.

P. S. C.

LOUIS ALEXANDER DUGAS, M.D., LL.D.

DR. LOUIS ALEXANDER DUGAS, one of the original members of the American Surgical Association, was born in Washington, Georgia, on January 3, 1806, and died in Augusta, Georgia, October 19, 1884, aged 78. His parents came to Georgia from St. Domingo, W. I. He attended medical lectures in Philadelphia and Baltimore, graduating in medicine at the medical department of the University of Maryland in 1827. After graduation, he visited Europe and spent several years in attendance upon courses of instruction in the schools and hospitals of the Continent. In 1832 he assisted in founding and organizing the Medical College of Georgia, in which institution he occupied the chair of surgery until a few years preceding his death, when he resigned, and was elected Emeritus Professor. He was a member of a number of literary and scientific societies,

among them the Geological Society of France, to which he was elected in 1834. He was President of the Medical Society of Augusta, of the Medical Association of Georgia, one of the Vice-Presidents of the International Medical Congress, held in Philadelphia, in 1876, and one of the Vice-Presidents of the American Surgical Association in 1881. The degree of LL.D. was conferred on him in 1869, by the University of Georgia. He was editor of the *Southern Medical and Surgical Journal* from 1851 to 1858, a period of seven years. He contributed to this journal and to other periodicals various articles upon medical and surgical subjects. Among these, the paper upon "New Diagnosis in Shoulder Dislocations" contributed most to his surgical reputation. In this paper he directed attention to an important diagnostic symptom in shoulder-joint luxations—the inability of the patient to touch the sound shoulder with the hand of the injured limb while the elbow rests in front of the chest.

He took an active interest in promoting the prosperity of the community in which he lived, and was esteemed, not only for his high professional attainments, but for his qualities as a good citizen. J. E. M.

JAMES LAWRENCE LITTLE, M.D.

Dr. James Lawrence Little was born in Brooklyn, New York, February 19, 1836, and died in New York, on April 4, 1885, in the 49th year of his age. He began his medical studies as a student of the late Professor Willard Parker and continued them at the College of Physicians and Surgeons of New York, where he graduated in 1860. He served as resident surgeon in the Bellevue and New York Hospitals. In 1863 he was appointed clinical assistant to Professor Parker in the College of Physicians and Surgeons, and in connection with his position, delivered a course of lectures in the spring term of the college. In 1868, on the organization of the regular summer faculty, he was appointed lecturer on operative surgery and surgical dressings.

He was elected to the chair of surgery in the University of Vermont in 1875. He was a member of a number of medical societies, among them the American Medical Association, the New York State Medical Society, the New York Academy of Medicine, and the Pathological Society of New York. In 1882 he was elected a Fellow of the American Surgical Association. His contributions to medical literature have consisted of papers read before the Societies of which he was a member, and of articles published in medical journals. The more important among these were papers upon the "Use of Plaster of Paris in Surgery," and "Median Lithotomy." Dr. Little was an able and industrious surgeon, giving much of his time to work in hospitals, to a number of which he was attached as attending surgeon. At the time of his death he was Professor of Surgery in the University of Vermont, and of Clinical and Operative Surgery in the Post-Graduate School of New York. J. E. M.

THE FIELD AND LIMITATION OF THE OPERATIVE SURGERY OF THE HUMAN BRAIN.

By JOHN B. ROBERTS, M.D.,
PROFESSOR OF ANATOMY AND SURGERY IN THE PHILADELPHIA POLYCLINIC.

NINE years ago, while resident surgeon in a well-known Philadelphia hospital, I watched a man die after a fracture of the cranium, and found at the autopsy, immediately under the fracture, a cerebral abscess from the surface of which was extracted a piece of bone driven in at the time of injury. I had seen the vigorous man admitted; seen him first intelligent, scarcely sick perhaps; then confused and delirious; then comatose, and finally dead. As the elements of this human life were being successively extinguished by increasing destruction of nerve-centres, the routine and approved medicinal treatment for encephalitis was steadily continued.

It took little surgical experience to recognize the lamentable result of the inactive, so-called conservative, treatment which almost to a certainty sacrificed the patient's life. Do we, Mr. President and Fellows of this Association, know no better at this day than to allow life to be thus destroyed through an almost criminal neglect of the most evident indications for surgical interference?

The recollection of this case and of similar ones has specially directed my attention to the importance of cerebral surgery; and it was with much pleasure that I accepted the President's appointment to prepare for this meeting a paper discussing the operative surgery of the human brain. The design of the paper is to indicate my views in such a succinct and categorical manner that the subsequent discussion may not be discur-

sive. The opinions and practice of the representative surgeons forming this body will thus, I trust, be so formulated that the profession at large may find in its TRANSACTIONS definite rules of action. Dr. Moses Gunn[1] and the present President, Dr. W. T. Briggs,[2] have, at previous meetings, given voice to their belief in more active surgical interference with pathological conditions of the brain. If their words had induced the profession to accept their conclusions, it is probable that I should not appear to-day to reiterate their thoughts.

Since I began the collection of material for this paper there has appeared the exhaustive article of my colleague, Dr. Charles B. Nancrede,[3] which leaves so little to be said, that I was almost inclined to lay aside my pen.

Perhaps, however, constant agitation of the topic may at last gain for those surgical principles which I am about to advocate a wider professional acceptance. It is possible, indeed, that I may take a position rather more radical than that which the authorities mentioned will accept as justifiable. During my remarks I shall report some illustrative cases, but promise that no great amount of time shall be consumed in the relation of their details.

It will, perhaps, facilitate discussion if I at once announce the opinions that I hold upon more or less disputed points involved in the consideration of my subject.

My creed, if I may use the term, is as follows:—

I. The complexus of symptoms, called "compression of the brain," is due not so much to displacing pressure exerted on the brain substance as it is to some form or degree of intracranial inflammation.

II. The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp

[1] Treatment of Fractures of the Skull, Recent and Chronic, with Depression. TRANS. AMER. SURG. ASSOC., Vol. I. p. 83.

[2] The Surgical Treatment of Epilepsy arising from Injuries of the Head, with special reference to the use of the Trephine, TRANS. AMER. SURG. ASSOC., Vol. II. p. 101.

[3] Injuries of the Head, International Encyclopædia of Surgery, vol. v. pp. 1–109.

is, with the improved methods of treating wounds, attended with very little increased risk to life.

III. The removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone.

IV. In the majority of cranial fractures the inner table is more extensively shattered and splintered than the outer table.

V. Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons.

VI. Drainage is more essential in wounds of the brain than in wounds of other structures.

VII. Many regions of the cerebral hemispheres of man may be incised and excised with comparative impunity.

VIII. Accidental or operative injuries to the cerebral membranes, meningeal arteries or venous sinuses should be treated as are similar lesions of similar structures in other localities.

IX. The results of the study of cerebral localization are more necessary to the conscientious surgeon than to the neurologist.

These propositions I shall consider successively:—

I. The complexus of symptoms called "compression of the brain" is due not so much to displacing pressure exerted on the brain-substance as it is to some form or degree of intracranial inflammation.

Many have argued against the use of the trephine in depressed cranial fracture, because considerable depression may exist without interference with the cerebral functions, and because "compression symptoms" frequently disappear after the lapse of a longer or shorter time, on account of the cerebro-spinal fluid, as they put it, gradually becoming displaced or absorbed, and the circulation being restored to its natural condition,[1] and because

[1] H. B. Sands, Annals of Anatomy and Surgery, vol. viii., 1883 p. 104.

deeper lesions beyond the reach of the trephine are frequently found in the fatal cases. Cure in these cases is supposed to occur by adaptation of the brain to the peripheral pressure. On the other hand, writers who favor more frequent operation have also clung to this compression theory, and have advocated perforation of the skull and elevation of the depressed bone for its relief. Their position has been assailed, and, I think, quite logically by those who use the line of argument mentioned above. Neither party seems to see that the so-called "compression symptoms," after traumatism, are not due to compression, but to some form or degree of inflammation. Let the profession repudiate the idea that brain displacement is the pathology of traumatic "compression of the brain," and many of the discordant opinions concerning the utility of trephining will be quickly harmonized. Some writers have, according to Stimson,[1] estimated that the constantly acting intracranial tension under normal circumstances is equal to from eight to twenty-five millimetres of mercury, and susceptible of temporary increase. This tension does, in some instances, overcome the displacement of the cranial vault. Duret,[2] after experimenting with injections of wax, states that a clot amounting to one-twelfth of the cranial capacity, situated between the dura mater and the bone, will cause coma and death in a few hours, but that in the arachnoid cavity a clot one-sixth or even one-fifth the bulk of the cranial cavity is requisite for a lethal effect. This results from the fact that, in the latter case, the wax moulds itself over a large surface instead of being confined to one spot.[3] Lesion of the nerve-centres must, of course, be absent. If this is true, how can any one believe that a square inch of bone, depressed a quarter or a half of an inch, will give sufficient pressure to cause cerebral symptoms, even if the presence of cerebro-spinal fluid does cause the external tension to be communicated to the entire cranial contents? Compression of the brain in cerebral tumors

[1] Treatise on Fractures, p. 251.

[2] Quoted by Nancrede in International Encyclopædia of Surgery, vol. v. p. 64.

[3] See Review of Études Expérimentales sur les Traumatismes Cérébraux, by H Duret, in Brain, vol. i., 1878, p. 106.

is the factor that causes many of the symptoms, but I am now speaking of surgical compression symptoms.

There are no definite symptoms by which we can distinguish accurately between laceration of the brain, contusion of the brain, and the so-called compression of the brain. Jonathan Hutchinson is almost correct in thinking that compression of the brain from depressed fragments of broken bone is an imaginary condition.[1] Briggs, with good reason, questions the view that secondary "compression symptoms" in intracranial suppuration are dependent exclusively upon the pressure of the pus. He suggests, and I agree with him, that the symptoms are more probably produced by the inflammatory process which caused the formation of pus.[2] Dr. Hunter McGuire,[3] on the other hand, said, at a recent meeting of this body, "If the fracture is a simple one, and the depression is not sufficient to bring on symptoms of compression, it is better to wait and see if the brain will not be able to accommodate itself to this condition." Here he admits a belief in the pressure theory, though in the next sentence he says that "we all know that there is no relation between the amount of depression and the symptoms of compression." At the same time, Dr. D. W. Yandell[4] gave as his opinion, that we should not elevate the depressed bone unless symptoms of compression are persistent. These opinions were given, I admit, several years ago, and the speakers may have changed their views by this time; but I am sure that to-day such words would still be spoken by many surgeons. The idea that depression of bone causes "compression symptoms" by mechanical pressure is deeply rooted. It would be well if traumatic "compression of the brain" were always translated "inflammation of the brain," and the profession taught to believe it due to irritation of the brain periphery from the traumatic cause.

II. The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp is,

[1] Statement of Briggs, Annals Anatomy and Surgery, vol. vii., 1883, p. 65.

[2] Loc. cit., p. 67.

[3] Transactions of the American Surgical Association, Vol. I. p. 92.

[4] Ibid., Vol. I. p. 97.

with the improved methods of treating wounds, attended with very little increased risk to life.

Uncertainty as to the character of a cranial lesion is more dangerous to health and life than the conversion of a closed into an open fracture of the skull, because observation has taught the profession that open cranial fractures do not resemble in fatality similar open fractures of long bones. If I but learn the character of the skull injury, I am acquainted with surgical expedients that render restoration to health more probable than the complication due to the incision renders it improbable.

Antiseptic methods have done away with much of the danger of open-bone wounds, as is shown by the frequent advocacy of resection for ununited and for malunited fractures, and of osteotomy and similar operations for conditions not urgently demanding surgical interference. No surgeon would hesitate to convert a closed recent fracture of the thigh or leg into an open one if it were otherwise impossible to replace fragments which were threatening life. Hence I strongly advocate exploratory incision of the scalp in obscure injuries of the skull.

Some months ago a man, who, it was said, had fallen from a heavy wagon, and had been run over, was admitted into my ward of St. Mary's Hospital. Behind the left ear was a large hæmatoma, where it was asserted the wagon-wheel had struck him. My resident surgeon made a two-inch incision, and removed the clots; but, finding no fracture, closed the wound with sutures, and applied corrosive sublimate dressing. The knowledge that no fracture existed was very satisfactory, I can assure you. Three days later, the incision had healed up without suppuration except in a space of three-eighths of an inch, and the sutures were removed. Such rapid union without complication will not always occur, but it shows the possibility of little risk in the majority of cases.

I recently ligated the brachial artery, and never dressed the wound after the hour of operation, because it was healed when the dressing was first removed. Such results of antiseptic surgery make us properly bolder than our predecessors, who feared to convert a closed fracture of the skull into an open one.

III. The removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone.

Much of the mortality attributed to trephining belongs to the serious brain-lesions that have accompanied the fractures for which trephining has been done, and to the absence of proper surgical antisepsis. Many patients have been trephined and have undoubtedly died; but the opponents of trephining must show that cause of death lay in the operation itself. In deaths occurring from lesions for which trephining is the admitted treatment, they must likewise show that the operation was done early enough to remove the causative factor of death before they can assert that the operation was unavailing. Gross, Michel, and many others, have frequently quoted historical facts and cases which show the slight risk incurred by uncomplicated trephining. Briggs says, "My opinion, based on a large personal experience, is, that trephining the skull is one of the safest of the capital operations of surgery."[1]

The opposite view, however, was held by Dr. H. F. Campbell, at a recent meeting of this Association, at which he said: "I have ever regarded trephining as one of the most serious of all capital operations."[2]

Nancrede,[3] from a careful investigation, gives a mortality of 10.69 per cent. as being a probably fair estimate of the risk of the operation *per se*, and a death-rate of 15.29 per cent. as an expression of the probable risk in trephining a simple depressed fracture. This author further says that his own experience has taught him that trephining is not a dangerous operation, and that more patients die from complications, that might have been prevented by timely operation, than from the removal of a disk of healthy bone.

Dr. R. W. Amidon, of New York, has collected 115 cases of trephining and kindred operations occurring since 1879. These

[1] Annals of Anatomy and Surgery, vol. vii , 1883, p. 65.

[2] TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, Vol. I. p. 94.

[3] International Encyclopædia of Surgery, vol. v. pp. 94, 95.

operations were done for various causes, and were unselected by Dr. Amidon; nor did he confine himself to cases treated antiseptically.[1] Of these 115 unselected cases, 29 died; but of these, 25 presented, at the time of operation, symptoms endangering life, leaving therefore but 4 cases in which the fatal issue could be attributed to the operation. This gives a mortality of a little over 3 per cent. to the operation. He announces his reasons for considering the 25 deaths as not attributable to operation in the following words: "In six cases, symptoms of abscess of the brain declared themselves before the operation was performed. In five a meningitis existed at the time of operation. In four cases shock caused death; two died of hemorrhage from a branch of the middle meningeal artery (not wounded in the operation); one died of hemorrhage from the middle cerebral artery, severed by a stab wound of the head; one died of hemorrhage from a lacerated longitudinal sinus; one of galloping consumption, which was hereditary; one of pneumonia; one of extensive laceration of the brain; one of opium poisoning; and three I accept, on authority of the physician reporting them, as not dying from the effects of the operation."[2]

Yeo[3] trephined 26 monkeys under antiseptic precautions, and had only one death attributable to intracranial inflammation, though six other deaths from exposure to cold weather, chloroform poisoning, or hemorrhage occurred among his cases. In some of the animals portions of the brain were excised. Of other monkeys trephined without antiseptic precautions all died.

Much stress has been laid by some writers on the danger of wounding the membranes and brain with the trephine. With a conical trephine or the burr of the surgical engine, as recommended by Dorr and by myself,[4] there is no danger of this.

[1] Annals of Surgery, St. Louis, March, 1885, p. 205; see, also, his previous paper, Medical News, June 21, 1884.

[2] This list accounts for the death of 26 instead of 25 patients, and apparently contains an error.—J. B. R.

[3] British Medical Journal, May 14, 1881, p. 763.

[4] Buffalo Medical and Surgical Journal, xix., 1879–80, p. 475; Phila. Med. Times, 1881–82, xii. 206.

Even if such an accident should occur to the membranes, it is, in my opinion, of very minor importance unless the damage is much greater than could occur except by gross carelessness. Dr. Gunn disapproves of the use of the mallet and chisel, because he believes that the repeated shocks to the brain may prove injurious to the nerve-tissues.[1] For removing large or irregular areas of bone, the flat burr of the surgical engine is certainly much more accurate and desirable.

I have compared trephining with amputation through the metacarpal bone, because in both operations there is exposure of cancellated bone structure. I do not know that the mortality of such finger amputations has been accurately computed, but it is certainly regarded by all as slight. It is seldom that patients are confined to the house after such amputations. Trephining in itself is, I am convinced, little if any more hazardous. I believe that one of us trephined to-day might, if it were necessary, go home without incurring any great risk to life; though I would not advise such a procedure. Amputation of the finger may be followed by erysipelas, septicæmia, or death; so may trephining, but it is not to be expected. The mortality of amputations of the thumb and fingers is, according to Ashhurst,[2] 3.3 per cent. This undoubtedly is less than the mortality of amputations through the metacarpus, because amputations of distal phalanges, which are almost without risk, are of course included. The same writer gives partial amputations of the hand a mortality of 6.6 per cent. If, therefore, we estimate amputation of a single finger through the metacarpal bone as having a mortality of 4 or 5 per cent., it will probably be nearly correct. According to the figures of Amidon, given above, trephining is actually much less dangerous to life than this.

IV. In the majority of cranial fractures, the inner table is more extensively shattered and splintered than the outer table.

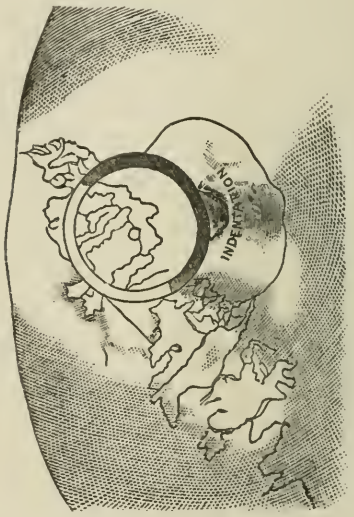
Many experimental fractures made in the dissecting-room, and observation of cases in the practice of myself and of others,

[1] Transactions of the American Surgical Association, Vol. I. p. 88.

[2] International Encyclopædia of Surgery, vol. i. p. 637.

teach me that extensive shattering of the inner table, with only a moderate amount of fracturing of the external table, is of frequent occurrence in other as well as in punctured fractures. I admit that the condition in the cadaver, preserved by zinc chloride, with its shrunken brain, is different from that in the living; but there is much evidence of the same splintering to be found in the study of accidental and homicidal cranial fractures. This is in accordance with the well-known mechanical law, that compressing force applied to the outside of a surface, as are undoubt-

Fig. 1.



Outer Surface of Fractured Skull.

edly most fracturing forces applied to the skull, tends to produce more extensive breaking of the inner surface. This is especially so in all localized blows. Punctured fractures have long been treated by early trephining, to avert encephalitis. For the same reason I recommend resort to trephining even in more diffused and less accentuated fractures. It is to prevent inflammatory sequences due to splinters forced into the membranes and brain, and to avert the consecutive occurrence of epilepsy and insanity, that the operation should be performed; not because of the

fear that symptoms of compression of the brain may arise, nor because necrosis of detached portions of bone may occur.

Sometimes there is no fissure in the outer table, though the inner table is extensively broken and depressed. Twenty such cases are reported as having occurred during the late civil war.[1] All of these patients died from intracranial inflammation except one, in which the splintered portion of the inner table was removed as a sequestrum.[2] I show you a piece of skull removed from a patient who recently died under my care.

Fig. 2.

Inner Surface of Fractured Skull.

He was struck with a pitcher, which caused a small scalp wound through which my finger-tip felt rough bone. I enlarged the incision, came upon a very rough surface, due to unusual irregularity of the lambdoidal suture, with small Wormian bones, and found only a small dent or fissure looking much like

[1] Med. and Surg. Hist'y Reb., Pt. I. Surgical Volume, p. 150.

[2] Prescott Hewett and Lidell have furnished other statistics of such cases. Holmes's System of Surgery, Am. edition, 1881, vol. i. p. 636.

the entrance for a vein. I determined to do exploratory trephining because of the nature of the vulnerating force. Dr. George Dock, under my direction, cut out a disk of bone close to the external dent, at the position which was thought would give best access to any splinters. Nothing was found but a small fissure crossing the inner surface of the disk. A probe slipped between the inner table and the dura disclosed no irregularity; therefore no further operative steps were taken. A portion of the inner table left in the bottom of the trephine-hole was undisturbed because it was smooth. The patient died in about forty-two hours of delirium tremens. Dr. H. F. Formad, the pathologist, found no inflammation of brain or meninges, but intense œdema of the brain and membranes, and at the inquest swore that death occurred from alcoholic delirium. The section of bone presented to you shows a marked depression of the table due to a T-shaped fracture under the seat of the external dent, beneath which was a small clot upon the dura mater. The top line of the T, which in the figure is vertical, is $1\frac{1}{4}$ inches long. The cleansed bone shows a semi-elliptical fissure of the external table encircling the slight dent. This was not seen at the time of operation, because the surface at that distance from the wound was left covered by periosteum. It would have been better if the trephine had been applied directly over the external dent instead of alongside of it. It is well, therefore, to follow that well-known rule in such cases as this, and also to employ a large trephine. Where a mere entrance for the elevator is desired, a small trephine should be used.

If this patient had lived, he would have been very liable, I think, to epilepsy or insanity. Only a few weeks ago I saw, in consultation with Dr. Charles K. Mills, a man of 22 years who was suffering with marked mental impairment occurring, as he and his brother said, subsequent to a fracture of the skull received about four years previously. Reference to the notes of the Pennsylvania Hospital, in which he had been treated, showed that he had been admitted for a compound depressed fracture of the skull, which was so slightly marked that operation was not deemed necessary. It is probable that his mental

impairment was due to a depression similar to that seen in this specimen. It was my intention to trephine in this case of mental failure if further investigation of his condition by Dr. Mills conclusively traced the aberration to the injury. Unfortunately, the patient passed out of our control by returning to his country home before I had a second opportunity of examination.

I feel sure that the element of danger in skull fractures is this splintering of the inner table; and I differ most decidedly from those who esteem it of comparatively little importance. Its danger in those abruptly depressed fractures, called punctured fractures, has been quite generally recognized; but its great frequency and risk in other forms of fracture are still not sufficiently emphasized by all authorities. Ashhurst, in the last edition of his Surgery, dated 1882,[1] says, in speaking of simple depressed fractures, "I have never seen a case of this kind in which I thought the use of the trephine justifiable, nor an autopsy which showed that the operation could possibly have saved life." In compound depressed *impacted* fractures he would not advise operation, "even if symptoms of compression were present." He goes on to say that the trephine "is not to be used with the idea of relieving compression, nor with the idea that there is any special virtue in the operation to prevent encephalitis." In punctured fracture the same distinguished surgeon thinks the trephine may be necessary to slightly enlarge the opening in the skull to remove the spicules which are apt to be broken from the more extensively involved internal table; but says, "It is better to leave imbedded in the brain, a foreign body, or even a fragment of bone, than to add to existing irritation by reckless attempts at its removal."

Perhaps Dr. Ashhurst has changed his views within the last two years. If he has not, I must disagree with him on this subject, except in the opinion that reckless surgery is always unjustifiable. His definition of what constitutes reckless surgery may differ from mine.

[1] Pp. 325, 326.

Many writers, while admitting the probability of greater damage to the inner table, do not give its disastrous consequences sufficient stress. Briggs,[1] however, truly says that the great danger in depressed fracture is not compression but inflammation set up by displaced fragments of bone. Dr. Sands says[2] that advocacy of early trephining, on the ground that loose pieces of bone in simple comminuted fracture will probably become necrosed and set up fatal intracranial inflammation, is improper. I believe that few advocates of early trephining give necrosis as a reason for their belief. Necrosis is not, but encephalitis is, very liable to occur.

Dr. Sands also believes that the apprehensions felt by those who advocate preventive trephining in closed depressed fracture without head symptoms, with the object of removing sharp fragments of the inner table, are scarcely justified by observation. These extensive osseous lesions are, in his opinion, often recovered from without surgical interference. He states[3] that immediate resort to the trephine is imperatively required, however, in fractures of limited extent, and in those in which there is reason to think, from their situation or the occurrence of monoplegia, monospasm, or hemiplegia, that a splinter has penetrated the motor area of the cortex. Trephining is also demanded, in Dr. Sands's opinion, when compression of the brain is due to blood between the dura and cranium.

V. Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons.

I have shown that the occurrence of fatal encephalitis is frequently due to spiculation of the inner table, and that spiculation or extensive shattering of the inner table is common in limited fractures of the external table. Hence it follows that exploratory perforation of the cranium is justifiable in all cases where the nature of the impinging force, or the appearance of

[1] Annals of Anatomy and Surgery, vol. vii. p. 69.
[2] Ibid., vol. viii. 1883, pp. 101–103. [3] Ibid., p. 106.

the external table, renders spiculation of the inner table probable, provided that less danger to life and health is inherent in perforation than in the probable spiculation. I have already asserted my belief in, and given reasons for, a low mortality risk of perforation. I am of the opinion that fractures of the cranial vault, produced by such general application of force as occurs when a man falls from a great height upon his head, are less frequent than fractures by direct and comparatively localized blows, such as occur from ordinary missiles, bullets, and falls from low elevations. These latter are those which tend to produce internal spiculation. Hence I am driven to the conclusion that exploratory perforation to determine the absence or presence of internal spiculation is often demanded by the uncertainty of the invisible condition. Without a knowledge of the true state of affairs treatment is empirical; and the risk to subsequent mental health or to life is too great to permit reliance on empirical treatment, when a knowledge of the true condition is obtainable with the slight danger that pertains to antiseptic trephining.

Whenever the fracture, whether originally an open one or so made by an incision, presents the possibility of the inner table being detached and splintered more extensively than the outer, I should be inclined to advise perforation. In other words, I would cut the scalp to see the condition of the outer table, and I would cut the bone to see the condition of the inner table, in every case where the risk of obscure knowledge is greater than the risk of divided scalp and perforated bone.

The tendency to procrastination in such matters has destroyed many lives. Nancrede[1] recommends early preventive trephining strongly, because, after encephalitis has once begun, trephining does not remove the inflammation, but merely one source of irritation without influencing the existence of the inflammatory process which has been aroused. His statistics show the mortality of the operation, after symptoms of brain disease have arisen, to be much more than twice as great as in preventive operations—the figures are 52.8 per cent. and 22 per cent. He

[1] International Encyclopædia of Surgery, vol. v. p. 95.

further says, that, although the operation should be done early, it is never too late in neglected cases to make the attempt; for an abscess may be found and evacuated with the result of saving life. Stimson[1] says that the percentage of recovery in early operative interference is actually high compared with tardy operations; and cites instructive cases to prove that his opinion is correct. Wound of the longitudinal sinus and removal of about three square inches of bone were no bar to recovery in a case which he treated by immediate trephining, though no brain symptoms except stunning were present.

VI. Drainage is more essential in wounds of the brain than in wounds of other structures.

That drainage is as essential in wounds of the brain as in wounds of other structures would seem to be a self-evident proposition; yet gunshot and other wounds of the skull and of the cerebral membranes and hemispheres have long been treated as if drainage was not a necessary factor in therapeutics. A surgeon who would think himself censurable to leave the opening leading into a suppurating cavity obstructed by shreds of skin and fascia, will, with profound equanimity, see his patient die with pus burrowing among the vital centres of the brain. There may be but a small fissure or opening, or perhaps none at all, in the bony wall inclosing the brain, and but a mere puncture in the tense dura mater, yet he will hesitate to give free exit to the pus. We split open a knee-joint, we incise a pleural, peritoneal, or pericardial cavity to give free egress of pus; so must we cut away the cranium and incise the dura mater in analogous conditions. The greatest error in this direction has, perhaps, been in an expectant, instead of an operative, treatment of bullet wounds of the brain.

Stimson,[2] in speaking of the trephine, says, "Most writers upon surgery during the last twenty or thirty years condemn its use unequivocally, except in compound fractures with depression and with marked and persistent cerebral symptoms."

[1] Treatise on Fractures, p. 248. [2] Ibid., p. 247.

A very few writers could be mentioned who are not included in the class spoken of by Stimson, but who have advocated more frequent use of the trephine in such skull fractures as are seen in civil practice. The hesitation to employ the trephine in bullet fractures, however, has been almost universal.

This is, I think, very erroneous doctrine, for death from abscess is very common after bullet wounds of the brain. The fracture is compound, the suppurative process recognized as almost certain to occur, and yet no provision of moment has, according to the usually pursued treatment, been made for drainage and antisepsis.

In a considerable number of open linear fractures of the cranium, pus has been found at the autopsy beneath the bone or membranes. The danger of an early trephining in such cases would have been less than that resulting from the imprisoned pus. The mere line of fracture establishes a direct communication between the air and the membranes which may lead to suppuration. Such communication is, of course, much more extensive in gunshot fractures and comminuted fractures. In cases, therefore, where there is reason to believe that suppuration will occur, trephining may be beneficial for purposes of drainage. I do not advocate perforation in all linear fractures which are not subcutaneous; but advise it when there is such strong probability of suppuration as is the case in nearly all gunshot fractures.

The statistics of Dr. H. R. Wharton,[1] concerning foreign bodies lodged in the brain, show the importance of removal when possible. Of 106 cases in which removal was accomplished only 34 died; of 210 cases in which no attempt at removal was made 122 died. The death-rate here was doubtless largely due to want of proper drainage.

While preparing this paper I assisted Dr. R. J. Levis in a case which will illustrate this phase of my communication.

A young man of nineteen years was shot in October, 1884, with a pistol of 22-hundredths calibre, held about a foot from his head.

[1] Philadelphia Medical Times, July 19, 1879.

The ball entered the skull at a point about 1½ inches behind and 2¼ inches above the cartilaginous attachment of the left ear, taking a direction slightly downward. He fell senseless, but soon reacted. The next day when seen he had no fever, no palsy, no strabismus, no abnormality of pupils, and no intellectual obscurity. Towards evening, however, his sight began to decrease, and by the morning of the second day after the accident he was totally blind, and had no pupillary reaction to light. Dr. A. H. McAdam asked Dr. Levis to assist him in the treatment of the case. A probe carefully inserted into the skull entered for a distance of about 3½ inches. The following afternoon (3d day) Dr. Levis trephined for the purpose of affording free drainage and of attempting extraction of the bullet, which he thought that he possibly had felt with the probe. An opening was made, the membranes incised, and several spicules of bone extracted from the cerebral hemisphere into which they had doubtless been driven by the ball. The brain-substance was already so broken down that it oozed out before the disk of bone was cut entirely loose, and the full length of my little finger was readily carried into the brain wound. The ball, not being readily found, was not searched for very long. On the next day (4th) the patient could distinguish the hand passed before his eyes. Two days later he could see to tell time approximately by a large clock hanging across the small room.

After the lapse of two more days, according to the notes of the case, he could tell the minutes indicated by the clock, which had a dial about ten inches in diameter, and was about six feet distant from him. Vision seemed better in the left than in the right eye, and when looking he closed the right eye as if some diplopia existed. Free suppuration from the wound continued, no marked febrile action occurred, and subsequently he became able to tell time by a watch. Nine weeks after the injury there occurred an abscess at the site of the trephining, but three months subsequent to the shooting he was attending to his business in good health, though the wound had not yet cicatrized. He could see well. I saw the patient only at the time of the operation, and have taken these facts from notes obtained by Dr. C. L. Bower.

I examined this patient in April, 1885, six and a half months after injury. He was in good health, weighed twenty pounds more than usual, drove a wagon, which he loaded with baskets and bundles, and had as good memory as ever for numbers, names, and words.

No failure in this respect was noticed by him in his occupation of delivering goods. He had no headache, and no tenderness or pain at the cicatrix of the scalp. I found that his left ear heard my watch tick at 24 inches, while his right heard it at 42 inches. This defect in hearing he had observed, and seemed to attribute it to the injury. He whistled well, but protruded the tongue with its tip a little to the left. No loss of tactile sense did I discover in testing sensation of the fingers with the points of a pair of compasses. He never observed trouble in vision before the injury, but said that now he could not read the newspaper well. The pupils reacted to light, and were neither dilated nor contracted. The right eye showed vision $\frac{20}{C}$, not perceptibly improved by spherical lenses or the stenopæic hole; the left eye $\frac{20}{LXX}$, not perceptibly improved by spherical lenses or the stenopæic hole. He read D 1.25 at $4\frac{1}{2}$ inches with difficulty, which convex lenses did not remove to any marked degree. No diplopia was noticed by him when looking at lights or stars. No strabismus, and no insufficiency of the internal rectus muscles were observed by me. There was, however, hemianopia of both eyes, when a pencil was held to the right of the middle line of each eye. The sense of smell in each nostril was apparently unaffected, for he easily distinguished carbolic acid from Cologne water. On each side of the tip of the tongue he readily distinguished tannin from chloride of ammonium. I regret that I had not a good opportunity of examining the ears and eyes with the ear mirror and ophthalmoscope respectively.

While admitting that a similar case might have recovered without operation I doubt its probability; the spicules driven into the brain, the softening therein already begun, and the profuse suppuration through the perforation, make me believe that death from intracranial abscess would have occurred rapidly if no operation had been done.

Agnew[1] quotes MacLeod as saying that in the Crimean War, of 67 gunshot fractures of the skull in which the skull was penetrated, one hundred per cent. died. Agnew himself gives a table[2] of 486 cases of penetrating gunshot fracture, with a mortality of 85.5 per cent. These high death-rates are largely,

[1] Principles and Practice of Surgery, vol. i. p. 287. [2] Ibid., p. 288.

I believe, due to inefficient drainage; but also to splintering of the inner table and driving of the splinters into the brain-substance. Dr. S. W. Gross is quoted by S. D. Gross as showing in over seven hundred cases of gunshot wounds of the skull only 25 + per cent. of recoveries by expectant or conservative treatment, but 41 + per cent. of recoveries after operative treatment; or a difference of about 16 per cent. in favor of operative interference. These statistics of Gross[1] were published eighteen years ago, and would probably be improved by the present operative methods.

Drainage of cerebral abscesses and wounds by tubes or horsehair has been successfully performed on several occasions. Noyes[2] passed a drainage-tube through a traumatic abscess in the frontal lobe, utilizing the trephine hole and the orbit for its entrance and exit. In this case death occurred. The four cases of similar operative treatment, reported by Burchard, by Fluhrer,[3] by Fenger and Lee,[4] and Kemper,[5] all recovered. Dr. J. P. Thomas,[6] of Kentucky, reports a case of cerebral abscess, which he successfully treated in 1875 by drainage obtained by position and the use of tents made of strands of silk.

These cases are sufficient to prove the truth of my sixth proposition.

VII. Many regions of the cerebral hemispheres of man may be incised and excised with comparative impunity.

Physiological experimenters know well that the lower animals bear, without serious risk to life, ablation of quite large areas of the brain-substance. Although clinical experience has shown the same fact respecting the human brain, surgeons have not

[1] System of Surgery, ed. 1882, vol. ii. p. 71, from American Journal of Medical Sciences, October, 1867.

[2] American Journal of Medical Sciences, July, 1882.

[3] Medical News, January 10, 1885, and New York Medical Journal, March 28, 1885.

[4] Transactions of the American Surgical Association, Vol. II., 1884, and American Journal of Medical Sciences, July, 1884.

[5] American Journal of Medical Sciences, January, 1885.

[6] Medical News, February 14, 1885.

been sufficiently impressed with the circumstance. They have looked upon the brain and its membranes very much as until recently they looked upon the heart and pericardium—regions never to be approached with trocar or scalpel. Many patients have died of brain abscess or brain tumor, because the timidity of the surgeon prevented the introduction of an aspirating needle or knife to open the pus cavity, or the employment of enucleation for the detachment of a neoplastic growth. This is partly due to the ignorance of surgeons respecting the localization of cerebral functions.

Nancrede[1] reports a remarkable case in which a patient, who was thought to be dead from cerebral abscess following penetration of the skull by a knife, was restored to conciousness by a second trephining and incision of the brain-substance. An aspirator needle had previously been passed several times into the brain to the depth of two-thirds of an inch through the first trephine hole, but no pus had been found. After the incision had evacuated one or two fluidounces of pus, the man revived and lived till the sixth day.

Dupuytren, Detmold, J. F. Weeds, Hulke, and others have reported recoveries after incision of the brain and evacuation of abscesses. Nancrede finds out of thirty cases of cerebral abscess treated by operation a mortality of only 50 per cent. I do not know in how many of these cases the brain-substance was incised or punctured in the search for pus.

Amidon[2] states, that of 100 cases of operation on the head, collected by him during recent years, the dura mater was opened in 33 by either the injury or the surgeon. These gave a mortality of 39.3 per cent.; but he states that in only one death could the fatal issue be directly ascribed to the exposure of the cerebral convolutions, and therefore he gives the mortality of operation inducing exposure of the hemispheres of the brain as 7.6 per cent. As showing the tolerance of the brain-substance to injury, he quotes among others the following recent cases, of which he gives the various sources as below mentioned.

Fluhrer's case. A bullet entering at a point about four centi-

[1] International Encyclopædia of Surgery, vol. v. p. 83; and TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, Vol. II., 1884.

[2] Medical News, June 21, 1884.

metres above the left eye, a little to the left of the median line, pierced the first frontal convolution, traversed the brain backwards and a little outwards, and emerged from the upper part of the parietal lobule, where it lay slightly imbedded in the brain. The symptoms were not alarming. The course of the bullet was ascertained by careful probing, and the trephine was applied over the supposed site of the ball. After a short search it was found, and a drainage apparatus composed of horsehair and catgut passed through the entire track of the ball. The operation was done with antiseptic precautions, and the wounds were dressed with iodoform. Recovery was uninterrupted and complete. The slight weakness of the right hand and loss of memory rapidly disappeared.[1]

Olpherts's case.[2] The whole of the right parietal, parts of the right occipital, temporal, and left parietal bones were torn away with extensive laceration and loss of cerebral substance. The case was treated antiseptically without operation, and recovered perfectly.

Wood's case.[3] A man was struck by the pilot of an engine, and his head cut open from the inner canthus of the left eye to the occiput. A furrow eight centimetres long was cut in the anterior lobe of the left hemisphere. Under cold applications he recovered.

Hoskins's case.[4] A boy six years of age was stepped on by a horse. The left side of the head was crushed in, blood, bone, and brain escaping. Blood and portions of brain exuded from left ear. It was estimated that 62 c.c. of cerebral substance were lost. There were three compound fractures. There was a right hemiplegia which disappeared, and an aphasia which improved.

These cases certainly prove the truth of my seventh proposition, which would also seem self-evident from the occasional continuation of life and even of comparative health during the existence of large areas of brain softening or suppuration, or of large substitutive growths in the cranial cavity.

[1] This case, quoted by Dr. Amidon with permission of Dr. Fluhrer, has since been published with most valuable remarks in the New York Medical Journal March 28, 1885.

[2] Olpherts, British Medical Journal, 1882, ii. 89.

[3] Wood, American Journal of Medical Sciences, July, 1881, lxxii. 168.

[4] Hoskins, Lancet, 1883, ii. p. 99.

VIII. Accidental or operative injuries to the cerebral membranes, meningeal arteries or venous sinuses, should be treated as are similar lesions of similar structures in other localities.

Numerous cases have recently been reported that prove this assertion. W. T. Bull[1] obtained good results in two cases after suturing the divided dura mater with catgut. Parkes[2] had a cure following lateral suture of the superior longitudinal sinus. W. B. Hopkins,[3] of Philadelphia, recently treated successfully a profuse hemorrhage from the superior longitudinal sinus, after removing comminuted bone, by pressure with a pad of lint sprinkled with iodoform. Sands[4] applied pressure to a wounded superior longitudinal sinus. Brinton[5] has applied lateral ligature successfully to the lateral sinus. Nancrede[6] has successfully ligated the middle meningeal artery. Numerous other cases of this sort might be cited, but enough have been quoted to prove my proposition.

IX. The results of the study of cerebral localization are more necessary to the conscientious surgeon than to the neurologist.

If any of the cerebral functions are with certainty recognized as located in definite convolutions and regions of the cortex of the cerebral hemispheres; if it has been demonstrated that perforation of the skull and incision of the membranes and brain will at times prevent the destruction of life by intracranial inflammation, abscess, or tumor; then it follows as a direct conclusion that a man, who assumes the rôle of a specialist competent to relieve disease by operative measures when medicinal therapeutics become impotent, must, if he is conscientious, make the subject of cerebral localization an object of study. I hold that such study is more necessary to him than to the neurologist, though the latter may, and probably does,

[1] Archives of Medicine, vol. i. 1879, p. 219.
[2] Annals of Anatomy and Surgery, viii. 1883. p. 118.
[3] Annals of Surgery, July, 1885.
[4] Annals of Anatomy and Surgery, vol. viii. 1883.
[5] Philadelphia Medical Times, vol. xii. 1881, p. 577.
[6] Ibid., March 8, 1884, vol. xlv. p. 440.

need a more detailed knowledge of the subject. The surgeon, when called upon to decide as to the propriety of operation, is often, and, indeed, usually, the sole authority by whom the question must be determined. If operation is required in these cases, it is required at once; no time can be wasted in waiting for the services of a neurologist living in a distant part of the county or State. Often, time to consult even books is scarcely available. Therefore, as many cerebral functions *have* been definitely localized, and many cases *have* shown that life can be saved by perforation of the skull and incision of the membranes and brain, I hold that the study of cerebral localization *is* more important to the conscientious surgeon than to the neurologist, who is seldom called upon to determine such questions in haste, except when the surgeon finds his own knowledge deficient.

Reference to a few of the cases in which cerebral localization, or functional cerebral topography, has been well utilized in surgical practice is here proper. Afterwards, I shall detail some of the facts which experimental and clinical investigation have proved to be of value to surgeons.

The recent removal by Godlee[1] of a gliomatous tumor the size of a walnut from beneath the gray matter of the upper part of the ascending frontal convolution, has probably attracted the notice of you all. The patient died after the expiration of four weeks from meningitis, which it is possible may have been due to inefficient drainage of the wound, as I have seen no statement that drainage was provided.

The chief symptoms which led Dr. Hughes Bennett, who had charge of the case, to diagnosticate the extent and locality of the tumor were paroxysmal twitchings of the left side of the face, alternating with twitchings of the left hand and arm, followed by slowly progressive paralysis of the hand, and later on by twitchings of the left leg and eyelid, and paresis of the left leg. These symptoms were accompanied by double optic

[1] Lancet, December 20, 1884, and January 3, 1885; see, also, Annals of Surgery, March, 1885, for Dr. Amidon's review of the case.

neuritis and violent headache. Dr. Bennett concluded that there was a brain tumor involving the cortical substance of the right hemisphere; that it was probably of limited size, as it had destroyed the centres presiding over motion of the hand, but had not yet destroyed the centres of the leg, face, and eyelids; and that to reach it, trephining should be done at the upper third of the fissure of Rolando. The operation was done, and the tumor readily removed. The lancinating pains in the head, the vomiting, the twitchings of the limbs were relieved; the temperature was, up to the twentieth day, never over 100°, and the pulse not above 90; and the patient was perfectly intelligent. Death occurred on the twenty-eighth day after rigor, fever, and pain had occurred during several days preceding.

It has been stated[1] that Macewen, of Glasgow, has twice successfully removed foreign growths, of which the site had been determined by the principles of cerebral localization. Other successful operations for localized purulent and hemorrhagic exudations, similarly detected, have been attributed to the same surgeon. He recently wrote me that he had not yet put the records of his cases in print, and that the accounts which had already appeared of some of them were not very trustworthy; but that he shortly intended to publish all of his cases bearing on this point. We are still, therefore, without any authoritative statement of the details of his operations. Broca, Lucas-Championnière, E. C. Seguin,[2] and E. C. Fuller[3] have given careful attention to, and valuable clinical information upon, the subject of cerebral topography in its surgical relations.

Dr. M. Allen Starr[4] has given a valuable analysis of American cases, in which there existed localized cortical lesions of the brain. Some of the cases are surgical, others medical, but his two papers deserve the careful perusal of every surgeon.

The following case of operation from the unpublished notes of Dr. Chas. K. Mills shows the value of cerebral localization:—

[1] Boston Medical and Surgical Journal, January 15, 1885.
[2] Archives of Medicine, December, 1882.
[3] Ibid., vol. ix. p. 262, 1884.
[4] American Journal of Medical Sciences, April and July, 1884.

Epilepsy following Cranial Traumatism; Situation of Meningeal Lesions diagnosticated by the Principles of Cerebral Localization; Trephining; Death.—A man, aged about 40 years, without any personal venereal history, and belonging to a family without any epileptic history, had, it was said, excellent health until eleven years before he came under Dr. Mills's observation. At that time, in 1873, he was struck upon the head many times with a boot-jack, and the mother was told by a physician that the skull was crushed, though the skin was not lacerated. Consciousness was not lost at the time of the injury. Great swelling of the parts followed immediately, and there occurred intense headache, which was almost constant, though much worse at times than at others. This was soon succeeded by attacks of vertigo, which sometimes resulted in his falling to the ground; consciousness, however, was retained. These attacks increased in severity until about three years after the receipt of injury, when he first began to suffer with true epileptic paroxysms, chiefly petit mal. The convulsions were slight or wanting, and unconsciousness lasted only a short time. The mother states that once a month he would have from three to six fits, after which he would be exempt from them until the next change of the moon.

The seizures gradually increased in severity and number during the eight years following his first paroxysm. During all these years he suffered with headache, which was always located at the seat of injury, which at times was so intense as to cause him to throw his hand to the seat of pain, and run and bury his head in his mother's lap.

About the time he first began to lose consciousness during the attacks (three years after injury) his eyesight rapidly began to fail, and he entered the Wills Eye Hospital, where he remained a short time. Previous to his death he was only able to see the name of the paper on a newspaper, and partly on this account, as well as on account of his headache, he seldom went out unattended by his mother. During the past weeks he appears to have suffered more than usual from his headache, which was described as a throbbing at the seat of the former injury, and his epileptic paroxysms came on him more frequently and were of a more convulsive character. His mother states that he would never bite his tongue during the convulsions, but that there would be some frothing at his mouth, and that his jaws would be fixed.

Five or six days before entering the Philadelphia Hospital, he

first noticed a weakness of the right arm, and a little later of the leg on the same side (the seat of injury being the left). About two days later he was unable to raise his arm, and in walking dragged his right leg. His mother does not know whether there was a disturbance of sensibility on the affected side or not.

His mother states that the convulsions followed each other at intervals of about half an hour, and that between them his mental condition had been the same as it was when Dr. Free, resident physician, found him on arriving in the ambulance. He was at that time lying on the floor, almost nude, in a maniacal condition, constantly repeating "I did not do it," all the while throwing his left hand up over the left side of his head, and then again down to his side or over his abdomen. His right arm and leg he did not move. The jolting of the ambulance seemed to promote his paroxysms, as he would have them every five minutes. On arriving at the hospital he was given 80 grs. of bromide of potassium, but with no effect. Nitrite of amyl was tried, but was found equally useless. Then inhalations of ether during the paroxysms, with hypodermic injections of morphia and atropia, were administered, which shortened the attacks. During the convulsion the left arm was in a state of rigidity and the fingers clinched. There was also marked clonic spasm of the arm and leg of the right side, alternating with some rigidity. The spasm in the right side differed from that in the left in being both clonic and tonic. The eyes were fixed, deviating to the right side, and the pupils very much dilated, the left slightly more so than the right; especially was the latter fact noticeable between the paroxysms. In the intervals his eyes showed a beautiful example of horizontal oscillation. This was bilateral, and the eyes acted together with perhaps a slight internal strabismus. Pulse very irregular, ranging from 100 to 160. His convulsions during the night followed each other at regular intervals of from 13 to 15 minutes, and if they were anticipated by inhalation of ether would be postponed until the end of the next 15 minutes.

About midnight he began to use his arms less wildly, his paroxysms occurred at longer intervals, and his pupils, which before had been widely dilated, contracted almost to pin-head size, but dilated during the paroxysms. Respiration was impeded or almost arrested after the convulsions, but never became stertorous. He passed very little urine. He became comatose, and remained so until June 5th, 5 P. M.

At this time the patient was first seen by Dr. Mills, and in consultation with Dr. Hearn it was decided to trephine. Although the mother stated that the injury was on the left side of the head, well forward, it was not indicated by any scar; but Drs. Mills and Hearn decided to trephine on the left, about the Rolandic line, concluding that the convulsions on the left were due to involvement of the dura mater, while those on the right to cortical irritation of the left hemisphere, in addition to the dural spasms.

The operation was done without the aid of an anæsthetic, and a piece of the skull removed. The dura mater was found rather more injected than was normal, and slightly adherent to the portion removed. No depression or fracture of the internal table was apparent. Soon after the operation he became quieter, his convulsions less marked, and he was again able to swallow and take a little nourishment during the night.

On the morning after the operation he spoke a few words intelligibly, and recognized his mother.

Nourishment was given him both by stomach and rectum, as he could retain but little in his stomach. His convulsions during the day and night were slight, and involved both sides. June 7th and 8th, second and third days after operation, it was evident that the man was becoming weaker, his pulse more feeble and rapid, but his temperature not much above normal; his breathing was hurried, but not stertorous, and he slept a considerable portion of the time. On the second day he had about six convulsions and but four on the third. On the night of June 8, 80 hours after the operation and four days and a half after beginning of his attack, the man died without a struggle, having had no convulsion during the night.

Autopsy held 13¾ hours after death.

Scalp wound the result of trephining, which had been performed June 6, 1884.

On the left side, at the seat of operation, the dura mater of convexity of left hemisphere, at a point one-fourth inch in front of fissure of Rolando and forward, was firmly adherent to pia mater and substance of the brain. The tissues as a whole formed a common mass. The dura mater was adherent to the skull over the anterior portion of this area, and was also very firmly adherent at base on left side under the middle lobe.

The trephining had been over the seat of meningitis. The dura

mater was much detached, particularly over the anterior frontal lobe, and adhered beneath to the gray and white matter.

Membranes showed necrobiotic spots.

Cause of death, pachymeningitis.

Although this dying man's life was not saved by operation, the seat of disease was determined by the symptoms, which was an important matter, since no scalp lesions or accurate clinical history pointed to the exact region of injury. Dr. Mills looks upon the case as one of meningeal disease induced by an injury, causing subcranial extravasation without fracture, which resulted in secondary meningitis, leading to adhesion. It is given here as an example of the value of localizing symptoms.

Enough is shown by these cases to prove the distinct surgical value of the study of cerebral localization, despite the opinion of one of our most distinguished members, who, in 1882, after speaking of the investigations of Broca and Lucas-Championnière, said, "I hope that I may be pardoned for expressing the opinion that their interesting investigations are more ingenious than practically useful."[1]

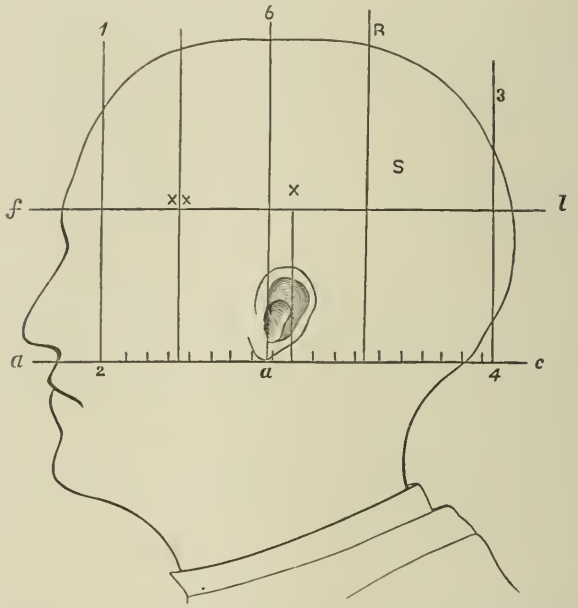
I shall therefore devote a little time to the discussion of the means by which the location of these cerebral convolutions, important to the surgeon, may be marked on the shaven head. The head must be so placed that a line drawn from the alveolar process of the upper jaw, at the base of the incisor teeth to the lowest part of the occipital bone, will be horizontal. Ranney[2] says this line or plane is easiest drawn in the living subject from the cusps of the teeth to the tip of the mastoid process. This alveolo-condyloid line is the base line from which other measurements are made. A perpendicular line drawn upwards from the alveolo-condyloid line through the external auditory meatus corresponds at the top of the skull with the transverse plane running through the bregma or point of intersection of the sagittal and coronal sutures. This line is called the auriculo-bregmatic line. The location of the bregma, which, of course,

[1] Ashhurst, Principles and Practice of Surgery, p. 326, note.

[2] Applied Anatomy of the Nervous System, p. 69.

lies in the median line of the head, is thus determined. Sometimes the bregma, which is the anterior fontanelle of infancy, can be felt through the scalp of adults; if it cannot be so distinguished, the ready methods of Broca or Lucas-Championnière may be

Fig. 3.

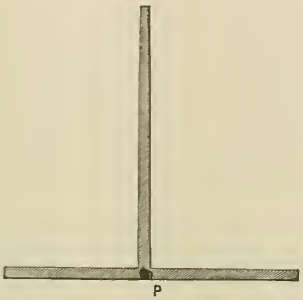


Outline of Head, reduced one-third, on which a modified system of Féré's lines is drawn (Amidon).—*a c*, alveolo-condyloid line; *a b*, auriculo-bregmatic line; *f l*, fronto-lambdoidal line; 1, 2, frontal limit; 3, 4, occipital limit of the brain; *R*, posterior extremity of the fissure of Rolando; *X*, anterior extremity of same; *X X*, location of the middle fold of the third frontal convolution, a little below and in front of which lies the tip of the sphenoidal lobe; *S*, the posterior extremity of the fissure of Sylvius. The intersection of the lines 3, 4, and *f l*, locates the occipto-parietal fissure.

employed instead of the mathematical one just described. A draughtsman's square of flexible material, such as steel, copper or tin, which has, at the intersection of the arms, a conical plug to go into the auditory meatus, may be used. The plug is to be placed in the ear, the horizontal arm bent until it lies across the upper lip directly under the columella of the nose, and the vertical arm then bent over the top of the head. The bregma is at the point where the axis of this vertical arm crosses the median line of

the skull. This probably will give the situation of the bregma more accurately than the original Broca square, which, according to Lucas-Championnière,[1] had the ear plug a little behind

Fig. 4.



Broca's Square.

the intersection of the arms. The other method of locating the bregma is to cut a section out of a sheet of pasteboard so that it will sit astride of the shaved head from auditory meatus to

Fig. 5.

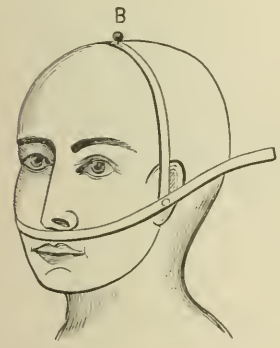


Fig. 6.

Methods of determining bregma.

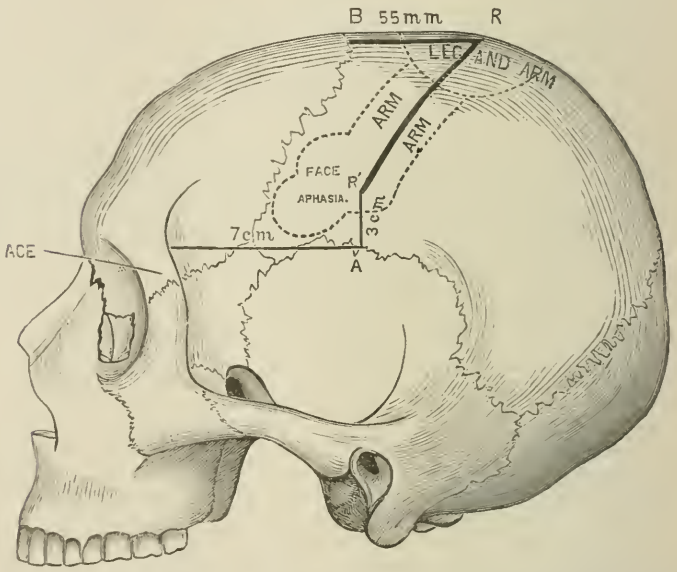
auditory meatus. In order to insure the pasteboard being perpendicular to the alveolo-condyloid line, a lead pencil or stick is thrust at a right angle through the pasteboard on a level with

[1] Trépanation guidée par les localisations cérébrales, p. 112.

the eyes. This enables the surgeon to detect any error in the horizontal position of the base line of the head. The centre of the arch in the cardboard is over the bregma.

The most important cerebral sulcus of each cerebral hemisphere concerned in our present studies is the fissure of Rolando, which has its upper end about 5 centimetres behind the bregma, but does not run quite up to the middle line. Some writers give 4.5 centimetres, others 5.5 centimetres. Its lower end lies about 0.5 centimetres behind the auriculo-bregmatic line, and a little

Fig. 7.



Diagram, from Nancrede, showing the method of locating on exterior of skull the fissure of Rolando, the motor areas of leg, arm, and face, and the centre whose destruction causes aphasia.

[If this is compared with my diagram on page 41 it will be seen that Nancrede gives distance of *R* from *B* as 5.5 mm., instead of 5 c., and marks leg and arm at top of motor area instead of leg alone I prefer the latter arrangement, but give his diagram here in addition to my own, which comes afterwards.—J. B. R.]

above a horizontal line, parallel to the alveolo-condyloid line, carried backwards from the superciliary ridge. This would place the lower end of Rolando's fissure about 6 centimetres above, and a little behind the external auditory canal. Another

method of fixing the lower end of the fissure of Rolando is to draw a horizontal line, that is a line parallel to the alveolo-condyloid line, from the external angular process of the frontal bone, where it begins to curve upwards to form the temporal ridge. At a point 7 centimetres distant from the anterior extremity of this line draw a perpendicular line extending upwards 3 centimetres. The end of this last line is over the lower end of the fissure of Rolando.

Erichsen quotes[1] Thane as follows: "The upper end of the sulcus of Rolando is placed about half an inch behind a point midway between the root of the nose and the external occipital protuberance; its lower end is close to the posterior limb, and about an inch behind the bifurcation of the fissure of Sylvius. The bifurcation of the fissure of Sylvius corresponds to a point one inch and a quarter behind and a quarter of an inch above the level of the external angular process of the frontal bone."

The occipito-parietal fissure corresponds nearly with the lambdoidal suture at the median line.

After the location of the fissure of Rolando has been marked, as with an aniline pencil, on the scalp, it is easy to locate the cerebral convolutions which control the movements of the opposite half of the head, body, and limbs.

"The first or superior frontal convolution will be found commencing about 2.5 centimetres behind the bregma, and passing forward near the median line toward the orbit. The second frontal convolution will occupy a similar but more lateral position, while the third frontal convolution lies wholly in front of the auriculo-bregmatic line, and on a plane 5 centimetres above the external auditory meatus. Its folded or central part is about 2.5 centimetres in front of the auriculo-bregmatic line, or about 2 centimetres behind the external angular process of the frontal bone."[2]

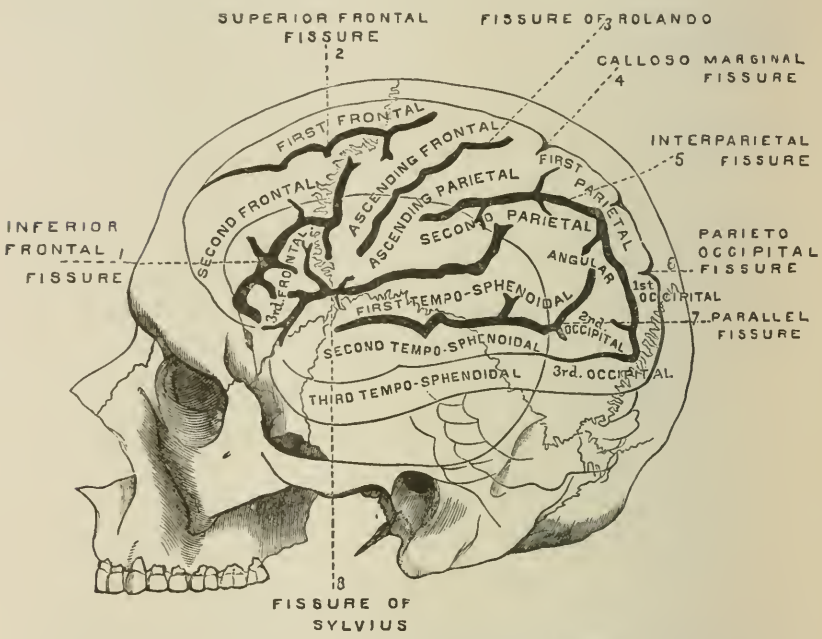
I have now shown how we can outline upon the scalp the situation of the cerebral convolutions. Hence, if we find symptoms which are known to be produced by lesions of cer-

[1] Science and Art of Surgery, i. p. 731.

[2] Amidon, Medical News, June 21, 1884.

tain regions of the cortex of the brain, it becomes our duty to trephine over the region indicated, provided that the symptoms are sufficiently particularized to indicate that a moderate, I do not say small, area of the brain is affected by disease.

Fig. 8.



Showing relation of the chief convolutions and fissures to the sutures and exterior of the skull (Diagrammatic).

Inflammation, abscess, or tumor, while circumscribed, will give a localized group of symptoms; soon, however, secondary œdema, inflammation, or softening may occur; and, by affecting other areas of the brain, will give additional symptoms. These secondary or indirect local symptoms will render accurate delineation of the seat of lesion difficult. Cases, therefore, should be studied accurately and carefully by competent observers at an early period, before the symptoms become more general through advancing disease, and the relief possibly derivable from operation diminished.

Cerebral localization is as valuable when informing us to abstain from operation as when telling that speedy operation is demanded. Loss of motion, or motor paralysis, is the symptom that has been chiefly depended upon as a localizing symptom in surgery, because it is more easily observed, and has been more carefully studied then loss of sensation, or sensory paralysis, and the other cerebral functions. The area about the fissure of Rolando is that in which the motor centres for the face and limbs have been found by physiologists and neurologists. It is necessary, therefore, to study these in more detail. As physiological knowledge increases, the sensory area of the brain will, in all probability, become equally important to surgeons.

I have formulated the most important facts according to recent physiological research, in order that the bearing of the corresponding surgical questions may be understood. The progress of knowledge may overthrow some of my conclusions, but that they are quite justified by present knowledge I think undoubted. The physiological portion and the tables which immediately follow, have been submitted to my colleague, Dr. Chas. K. Mills, who, while not responsible for my statements, has kindly given me valuable suggestions. In order to use these facts a general diagnosis of a cortical lesion, that is, of a lesion not of the ganglia or tracts, must first be made.

Motion.—The gray matter of the ascending frontal and ascending parietal convolutions, which are separated from each other by the fissure of Rolando and coalesce over the top of this fissure in the paracentral lobule, is the portion in which are chiefly located the motor centres for the muscles of the face, tongue, arm, and leg of the opposite side of the body, although the motor zone may extend forwards and backwards slightly beyond the strict limits of these convolutions. Some few nerve-fibres are in relation with the muscles of the same side, but nevertheless, in fully 90 per cent. of lesions of the cortical centres the anomalies of motion, namely, spasm and paralysis, occur on the side opposite that on which the brain lesion exists. This statement is subject to modification to be presented when speaking of the rôle of the dura mater. These ascending convolutions are conveniently spoken of as the central region.

The motor area for—

Face and *tongue* lies in central region, *lower third.*
Arm " " " *middle* "
Leg " " " *upper* "

In these areas are the centres for the individual muscles of said limbs, face, and tongue.

Spasm of one of these groups of muscles indicates irritation of the corresponding motor area in the brain cortex.

Paralysis of the same group of muscles indicates destruction of the same motor area.

Spasm or paralysis of two of these groups indicates that the lesion involves more than one area, for example:—

Paralysis of one side of the face and of the corresponding arm indicates a destructive lesion of the lower and middle thirds of the central region.

Paralysis of the arm and corresponding lower limb points to a destructive lesion of the middle and upper thirds of the central region.

Spasm would indicate similarly located irritative lesions.

Paralysis of face, arm, and leg (hemiplegia), would indicate destructive lesion of the lower, middle, and upper thirds of the central region; and spasm of the same muscle, irritative lesion of all three portions of the central region; except that it must be remembered that a primary lesion of one area sometimes affects secondarily the neighboring area or areas and causes hemiplegia.

This is explained by several facts, among which are these—

1. The blood supply to the central region comes entirely from the middle cerebral artery, which ascends in the pia mater upon the surface of the convolutions from the base in the vicinity of the island of Reil. Hence, a lesion in a lower area may cause secondary symptoms in the area or areas above by cutting off its arterial supply.

2. The nerve-fibres converge as they pass inwards from the cortex to make up the bundles traversing the interior of the brain in their journey to the muscles. Hence, a lesion whose influence extends by pressure or otherwise below the surface a short distance may implicate converging fibres, coming from surround-

ing cortical areas, and thus give results such as would occur if these more distant cortical areas were also really involved.

These and other facts explain the occurrence, in some cases, of hemispasm or hemiplegia, or of extensive spasm or extensive paralysis, from limited lesions. If the lesion be a small focus of disease, thus giving rise to extensive spasm or paralysis, it usually will first cause symptoms in the group of muscles supplied from the small area primarily affected, or the symptoms in that group will be more marked than in those secondarily involved. This point is of diagnostic importance. Hence the relative time of occurrence of symptoms always demands careful investigation. When monospasm or hemispasm is one of the consensus of symptoms, the seat of the primary lesion may often be determined by study of character of that spasm, especially by attention to the special muscles in which the spasm began. It is likely to be in that part of the cortex which is the area of control for this special group of muscles.

Paralysis of groups of muscles following spasm of the same is characteristic of disease of the central region.

Seguin suggests that a latent hemiplegic state may be discovered, at least in some cases, by an increase of peripheral temperature, as of fingers or toes, on one side, or by the presence of congestion, or erythema, on one buttock.[1] This may be valuable in diagnosticating cerebral hemorrhage from alcoholic coma and similar affections.

Sensation.—Late observation and experiment have proved that disturbance of general sensation also is one of the direct local symptoms of lesions in the central region. These disturb ances include the senses of touch, pressure, pain, and temperature, and the sense of location of a limb, and may consist in impairment of these functions or the perception of such sensations when there is no objective cause to produce them (hallucinations of sense). General sensation then is located in the central region, and especially it would seem in the portion behind the fissure of Rolando, that is, the ascending parietal convolu-

[1] Gross, System of Surgery, vol. ii. p. 44.

tion. It must not be forgotten, however, that the sensory region seems to include also the parietal lobes which lie behind the ascending parietal convolution, and which it will be observed contain but few motor centres and fibres. The motor and sensory regions thus seem to overlap; for lesions in front of the fissure of Rolando have caused sensory as well as the usual motor disturbances; and we have seen that motor disturbances may be due to lesion behind the fissure of Rolando in the ascending parietal convolution where I have just said sensory areas exist. It is possible that future and more accurate investigation will show that the motor region is particularly located in front of the fissure of Rolando, but also extends on the posterior side of the fissure, overlapping, or fading into, the sensory region behind the fissure, in a manner similar to that in which the motor areas for the arm and leg appear to overlap or fade into each other. It is more probable, perhaps, that we have not yet sufficiently defined the exact boundaries, because our methods of investigation are not sufficiently accurate. As is the case with the motor centres, which are connected especially with the opposite side of the body, so is it with the sensory centres; though a few sensory fibres go, as do a few motor fibres, to the same side of the body. Hence, sensory lesions give more marked sensory disturbance on the side opposite the lesion, and affect in a less degree the side corresponding to the lesion.

The sensory area for—

Face probably lies in the central region, *lower third.*
Arm " " " " " *middle* "
Leg " " " " " *upper* "

The area in each case probably extends also behind the fissure of Rolando in the ascending parietal and parietal convolutions.

Hyperæsthesia, pain, or paræsthesia of the face, arm, or leg, indicates irritation of the corresponding sensory area in the brain cortex.

Anæsthesia and analgesia indicate destruction of the corresponding sensory area in the brain cortex.

Hyperæsthesia or anæsthesia of two cutaneous tracts indicates that the lesion involves more than one area. For example:—

Anæsthesia of one side of the face and of the corresponding arm indicates a destructive lesion of the lower and middle third of the central region, probably situated behind the fissure of Rolando, and also in the parietal convolutions.

Anæsthesia of the arm and corresponding lower limb points to a destructive lesion of the middle and upper third of the central region, probably situated behind the fissure of Rolando, and also in the parietal convolutions.

Hyperæsthesia would indicate similarly located irritative lesions.

Anæsthesia of face, arm, and leg (hemianæsthesiä), would indicate destructive lesion of the lower, middle, and upper thirds of the central region, probably situated behind the fissure of Rolando, and in the parietal convolutions; and hyperæsthesia of the same cutaneous tracts irritative lesion of all three portions of the central region, probably situated behind the fissure of Rolando, and in the parietal convolutions.

If motor paralysis is associated with these sensory disturbances in a cortical lesion, the probability is, that we have a lesion involving both the ascending and the parietal convolutions. If the motor paralysis is slight, the lesion probably predominates in the parietal convolutions.

Loss of the Special Senses.—In localizing brain lesions by impairment of the special senses it is important to eliminate all lesions occurring between the cortical centre and the sense organ. For example: loss of sight in brain disease may be due to an increase of intracranial pressure from a tumor causing choked disk; or loss of smell to pressure of a growth on the olfactory nerves after they leave the base of the brain. Such sources of error must be eliminated. This often requires careful study of the case.

Hemianopia, failure to recognize or remember familiar objects, or hallucination of vision, indicates disease probably in the occipital lobes, not far from the angular convolution. If the failure of sight includes the whole field of vision, both hemispheres are involved; if hemianopia only is present, one hemisphere is involved. Blindness of the right half of both eyes, that is, oblitera-

tion of the right half of the field of vision, indicates destructive lesion of the left occipital lobe, though it is the left half of the retina which is impaired.

Hemianopia on the left side means lesion of the right side of the brain.

Deafness in one ear or hallucinations of hearing on one side (such as voices and music) indicate disease of the first temporo-sphenoidal convolution of the opposite side. Failure to recognize or remember spoken language indicates lesion of the left first temporo-sphenoidal convolution in right-handed persons and of the right first temporo-sphenoidal convolution in left-handed persons.

Loss or hallucination of smell possibly indicates disease at the base of the brain in the temporo-sphenoidal region, which could not readily be reached by surgical procedures.

Loss of taste has not been sufficiently studied to afford even the suggestive evidence that we possess about disturbances of smell.

Disturbance of other Conscious Mental Actions, such as judgment, reason and self-control, will be exhibited in changed disposition and character of the patient. Such change, if no other general or localizing symptoms exist, indicates disease of the frontal lobes.

Aphasia.—Disturbance in speech power indicates disease in the neighborhood of the fissure of Sylvius on the left side in right-handed persons; on the right side in left-handed persons. It must be recollected that neurologists distinguish three kinds of aphasia.

Aphasia may be due to loss of—

1. Memory of words; whose centre is situated in the first temporo-sphenoidal convolution. (Sensory aphasia.)
2. Memory of voluntary acts necessary to speech; whose centre is situated in the posterior part of third frontal convolution and lower part of central region (ascending frontal convolution). (Motor aphasia.)
3. Connection between the regions just described; which is required to turn thought into speech. This function is located in fibres passing through island of Reil and lower part of central region (ascending frontal convolution). (Aphasia of incoördination.)

There is some difference of opinion between French and German writers as to the distribution of these centres, but for our surgical purposes the assertions above given are sufficiently accurate. It will be seen that the convolutions mentioned are in close anatomical relation. I have followed on this point and in many other respects the elaborate *résumé* of Dr. Starr, who has analyzed a large number of reported American cases.[1]

Fig. 9.

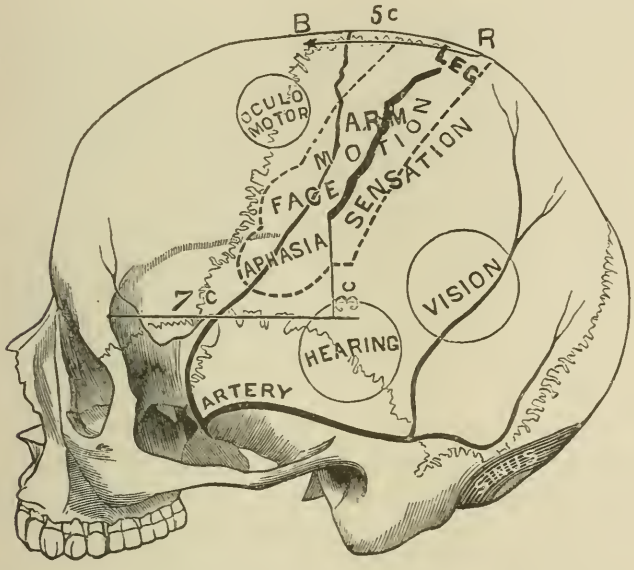


Diagram of the Exterior of the Skull.—Upon this have been indicated the measurements for determining the position of the fissure of Rolando; and the location of the regions under which lie the centres of motion and sensation, as well as those of vision and hearing, and the oculo-motor centre. The position of the grooves for the two branches of the middle meningeal artery and for the lateral sinus is also shown.

If the patient understands a question and can recall the words for a reply but cannot make the requisite speech movements he has motor aphasia, which indicates disease in the third frontal and lower end of the ascending frontal convolutions.

[1] Cortical Lesions of the Brain, by M. Allen Starr, Am. Journ. Med. Sciences, April and July, 1884.

If he cannot recognize spoken language, but is able to repeat words after another person, or can use exclamations on being annoyed he has sensory aphasia, which indicates disease in the first temporo-sphenoidal convolution.

If he can understand and talk, but puts an unexpected word for the one desired, he has aphasia of incoördination, which indicates disease situated deeply within the Sylvian fissure or in the island of Reil and the white substance thereabouts, and involving the association fibres.

These points need further study, I admit, because all authorities do not exactly concur in these locations; but the difference is not enough to disturb the question of operation. When opening the skull in the search for abscess or tumor located by localizing symptoms the surgeon should use a large trephine, because the exact spot of lesion is not determinable.

From a study of the points just discussed and the writings of Lucas-Championnière[1] and A. L. Ranney[2] I present the following deductions concerning the proper points for operation, when lesions amenable to operative treatment have been distinguished and localized by the symptoms exhibited. It must be remembered, however, that I am treading very often on debatable ground, and have made statements which from increase in knowledge may require modification in a few weeks or months. I have endeavored to be categorical, and at the same time to indicate by my phraseology those opinions and logical positions which are most satisfactory and certainly proved to be correct.

The researches of H. Duret,[3] who has shown that some motor symptoms may be due to traumatic irritation of the dura mater, must not be forgotten in studying individual cases. The non correspondence of the symptoms, observed in a given case, with the typical symptoms described as accompanying a lesion in that locality, may be due to a dural lesion, in addition to the cortical lesion; and, on the other hand, certain observed symp-

[1] Trépanation guidée par les localisations cérébrales.

[2] Applied Anatomy of the Nervous System.

[3] On the Role of the Dura Mater and its Nerves in Cerebral Traumatism. See *Brain* (1878), vol. i. p. 29.

toms may be caused by dural rather than cortical lesions. These questions are too intricate to be further discussed at this time, but must be indicated by me, in order that I may not deceive you into believing that cerebral localization is an easy problem in all cases of brain injury or surgical disease.

Duret says the dura mater contains sensory nerves eminently excitable, and that

"I. Irritative lesions of these nerves cause,

"1. Pain, hyperæsthesia, neuralgia, and reflex motor phenomena.

"2. Reflex spasms or contractures of the muscles of animal and organic life.

"(*a*) The spasms or contractures of the muscles of animal life may occur in the face, eyeballs, neck, trunk, or limbs. They occur sometimes on one side, sometimes on the other." [This is to us an important item.] "These symptoms tend to diffuse and invade neighboring groups of muscles. They have never the localization, the measured and purposed character of the contractions which belong to the lesions of the cortex. They frequently become transformed into permanent contractures.

"(*b*) The reflex vasomotor disturbances, due to irritation of the nerves of the dura mater, consist in spasms or congestive paralysis of the cerebral and ocular vessels, either on the same or the opposite side.

"These facts are important to pathologists, as they show the great influence of irritation of the nerves of the dura mater on cerebral vascular conditions, and on the organs of sense, and on the causation of secondary effects in cerebral traumatism, *i. e.*, on the congestions and inflammations of the cerebral membranes.

"II. Destructive lesions cause local anæsthesia of the dura mater."

I have made this long quotation from Duret because of its importance.

TABLE I.—INDICATIONS IN TRAUMATIC CASES.[1]

1. Hemispasm, or 2. Incomplete hemiplegia, or 3. Hemispasm with hemiplegia,	on the opposite side of the body after even slight injury in the fronto-parietal region, and even when not directly over the motor area,	indicates exploratory operation at the site of injury.
4. Monospasm, or 5. Monoplegia, whether total or incomplete,	do. do. do. do.	indicates, indeed demands, exploratory operation at the site of the injury.
6. Hemihyperæsthesia, or 7. Hemianæsthesia, or 8. Hemianalgesia, or 9. Hemihyperæsthesia with hemianæsthesia or hemianalgesia,	on the opposite side of the body after even slight injury in the parieto-occipital region near or somewhat behind the motor area,	may indicate exploratory operation at the site of injury.
10. Monohyperæsthesia, or 11. Monoanæsthesia, or 12. Monoanalgesia,	do. do. do. do.	may indicate, or may perhaps demand exploratory operation at the site of injury.
The 12 symptoms, above mentioned, if on the same side as the injury, contra-indicate operation at the site of the injury, but		may indicate exploratory operation on the side of the cranium opposite the injury.
13. Coma, with Hemispasm, or Hemiplegia,	in cases of supposed subcranial or arachnoid hemorrhage,	may indicate exploratory operation on the side opposite the paralysis or spasm.
14. Coma, with Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia,	in cases of supposed subcranial or arachnoid hemorrhage,	may indicate exploratory operation on the side opposite the hyperæsthesia, anæsthesia, or analgesia.

[1] See note on dura mater and its lesions, p. 43.

Table II.—Contra-indications.

1. Paralysis of one or more cranial nerves, Neuro-retinitis, Cheyne-Stokes respiration, or other symptoms showing lesions probably at the inaccessible base of the brain, or in undeterminable parts of its interior,	even if other symptoms indicate operation are, except in cases of supposed cerebral abscess,	contra indications to exploratory operation.
2. Complete hemiplegia, which often is not of cortical origin, but is due to lesions of deep structures,	is, except when— 1, it is due to a supposed cerebral abscess, 2, it has been incomplete at outset, 3, it is irregular, and corresponds with extensive depression of bone on opposite side of cranium,	a contra-indication to exploratory operation.
3. Hemispasm, or Hemiplegia, with marked hyperæsthesia or anæsthesia, indicates more extensive cortical lesion than hemispasm or hemiplegia alone, and may even be due to deep lesions, and	is, except when— 1, the hyperæsthesia or anæsthesia is limited to a small cutaneous surface (monæsthesia) 2, the hyperæsthesia or anæsthesia is apparently due to the same cortical lesion that causes the spasm or paralysis,	a contra-indication to exploratory operation.
4. Hemispasm, or Hemiplegia on same side as injury,	may, if other symptoms indicate operation, be an indication for operation on the side of the cranium opposite to that of the injury, but	is a contra-indication to exploratory operation on the injured side of the cranium.
5. Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia, with marked spasm or paralysis, indicates more extensive cortical lesion than hemihyperæsthesia or hemianæsthesia, hemianalgesia alone, and may even be due to deep lesions, and	is, except when— 1, the spasm or paralysis is limited to a small group of muscles (monoplegia), 2, the spasm or paralysis is apparently due to the same cortical lesion that causes the hyperæsthesia or anæsthesia,	a contra-indication to exploratory operation.
6. Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia, on same side as injury,	may, if other symptoms indicate operation, be an indication for operation on the side of the cranium opposite to that of the injury, but	is a contra indication to exploratory operation on the injured side of the cranium.

Note.—When the symptoms indicate that many centres are involved, and this is usually the case, more bone must be removed than by a single trephine perforation. The burr of the surgical engine, or the gnawing bone forceps, is the most convenient instrument for enlarging the trephine hole.

TABLE III.—POINTS FOR OPENING THE CRANIUM WHEN OPERATION IS INDICATED.

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.	Remarks.
Hemispasm or hemiplegia.	Along entire length of fissure of Rolando, or possibly along a portion of its extent, and involving other areas secondarily, more probably in front rather than behind fissure.	Over middle of fissure of Rolando, and to be extended downwards, upwards, and forwards in search for lesions, rather than backwards, unless disturbances of sensation have been present; then backward extension is indicated. If aphasia is present the opening should be extended downwards and forwards.	
Crural monospasm or monoplegia (convulsions or paralysis limited to one leg, or part of one leg).	Upper third of central region in ascending convolutions and astride of upper end of fissure of Rolando. Possibly in front rather than behind fissure (?). Some say in ascending parietal convolution rather than in frontal (see Erichsen, i. p. 725, and Lucas-Championnière).	Over upper end of fissure of Rolando, and to be extended forwards rather than backwards, unless disturbance of sensation is present.	
Spasm or paralysis of both legs; not due to spinal lesion.	Upper part of central region, and paracentral lobules on both sides.	Over middle line between upper ends of both fissures of Rolando, to be extended towards the side of vault of cranium which is opposite to the side of the more pronounced symptoms. Take care to avoid if possible injury to superior longitudinal sinus.	
Brachial monospasm, monoplegia (convulsions or paralysis limited to one arm, or part of one arm).	Middle third of central region in ascending convolutions, especially in ascending frontal.	Over middle third of fissure of Rolando, but rather more in front than directly over the fissure, though the fissure should be uncovered. To be extended forwards rather than backwards, unless disturbance of sensation is present.	Facial spasm or paralysis is often associated with the crural symptoms; as by hemorrhage gravitating downwards, etc.
Associated crural and brachial monospasm or monoplegia.	Upper two-thirds or at junction of upper and middle thirds of central region, probably in ascending frontal rather than ascending parietal convolution.	Over junction of upper and middle thirds of fissure of Rolando.	

TABLE III.—*Continued.*

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.	Remarks.
Facial spasm or paralysis.	Lower third of central region, especially in ascending frontal and posterior end of second frontal convolutions. The anterior part of this area seems to control chiefly the motions of the upper part of the face, and the posterior part the motions of the lips or mouth.	In front of lower third of fissure of Rolando, though the posterior edge of opening should be close to the line of the fissure of Rolando.	Aphasia is often associated with the facial symptoms.
Aphasia.	Below and in front of lower end of fissure of Rolando, varying somewhat with form of aphasia. 1. Sensory aphasia in first temporo-sphenoidal convolution. 2. Motor aphasia in posterior part of third frontal and lower part of ascending frontal convolutions. 3. Aphasia of incoördination in vicinity of island of Reil. Aphasia in right-handed persons is due to lesion on left side of brain; in left-handed persons to lesion on right side of brain.	About a half inch (1.25 centimetres) below, and a half inch (1.25 centimetres) in front of the lower end of the fissure of Rolando.	Single large opening will cover the convolutions for the three forms of aphasia, but the area for the third form is deeply situated in the fissure of Sylvius.
Associated brachial and facial spasm or paralysis.	Convolutions involved are those of arm and face as given above.	In front of junction of middle and lower thirds of fissure of Rolando.	
Associated brachial and facial spasm or paralysis with aphasia.	Convolutions involved are those of arm, face, and speech as given above.	In front of lower third of fissure of Rolando, and to be extended upwards or downwards and forwards according as brachial or aphasic symptoms are more pronounced.	
Associated facial spasm, or paralysis and aphasia.	Convolutions involved are those of face and speech as given above.	About a half inch (1.25 centimetres) directly in front of the lower end of the fissure of Rolando, and to be extended upwards and backwards, or downwards and forwards, according as facial or aphasic symptoms are more pronounced.	

Table IV.—More Uncertain Localizations.

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.
Hemihyperæsthesia, Hemianæsthesia, Hemianalgesia.	Behind fissure of Rolando in ascending parietal and first and second parietal convolutions.	Behind the middle of fissure of Rolando, and to be extended backwards rather than forwards.
Crural monohyperæsthesia, Crural monoanæsthesia, Crural monoanalgesia.	Upper third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind upper third of fissure of Rolando.
Hyperæsthesia of both legs, Anæsthesia of both legs, Analgesia of both legs. Not due to spinal lesion.	Same region as case above, but on both sides of the brain.	Over middle line behind the upper ends of both fissures of Rolando, to be extended towards the side of the vault of cranium opposite to the more pronounced symptoms. Take care to avoid if possible injury to superior longitudinal sinus.
Brachial monohyperæsthesia, Brachial monoanæsthesia, Brachial monoanalgesia.	In middle third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind middle third of fissure of Rolando.
Associated crural and brachial Monohyperæsthesia, Monoanæsthesia, Monoanalgesia.	Upper two-thirds of ascending parietal convolution and portion of parietal convolutions just behind.	Behind middle two-thirds of fissure of Rolando.
Facial hyperæsthesia, Facial anæsthesia, Facial analgesia.	Lower third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind lower third of fissure of Rolando.
Associated brachial and facial Monohyperæsthesia, Monoanæsthesia, Monoanalgesia.	Lower two-third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind lower two-thirds of fissure of Rolando.

TABLE IV.—*Continued.*

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.
Loss or change in power of attention, reason, judgment, or self-control.	Frontal lobes.	Surface of frontal bone, but the spot could only be located by some other symptoms which might be present, such as aphasia ocular deviation, affections of the leg or arm.
Muscular deviations of the eye and dilatation of the pupil.	In first and part of the second frontal convolutions in front of motor centre for arm, reaching nearly to the longitudinal fissure.	About three centimetres in front of spot for exposing motor centre of arm.
Blindness or failure to recognize and remember familiar objects, or hallucinations of vision.	Occipital lobes near parietal lobes, not far from angular convolution. Blindness of half field of both eyes at *right* side of body indicates destruction of *left* lobe, and vice versa.	About seven centimetres above the external occipital protuberance, and seven centimetres around the skull laterally.
Deafness, or hallucinations of hearing.	First temporo-sphenoid convolution of side opposite to affected ear. Failure to recognize or remember *spoken* language is a form of aphasia (sensory aphasia), and is due to lesion in first temporo-sphenoidal convolution.	Just below and about three centimetres behind the lower end of the fissure of Rolando.
Loss or hallucinations of smell.	Probably at base of brain in temporo-sphenoidal region.	Not accessible to operation.
Loss or hallucinations of taste.	Not located.	

I have now with great detail discussed what I conceive to be the principles of the operative surgery of the human brain. It remains to apply these principles to the treatment of—

A. Cranial fractures.
B. Intracranial hemorrhage.
C. Intracranial suppuration.
D. Epilepsy following cranial injury.
E. Insanity following cranial injury.
F. Cerebral tumor.

A. In discussing the treatment of cranial fractures I shall ask myself four questions:—

1. What conditions demand incision of the scalp?
2. What conditions render incision of the scalp unjustifiable?
3. What conditions demand perforation of the skull?
4. What conditions render perforation of the skull unjustifiable?

These queries are best answered by the tabulated statement which follows. I admit that the line of treatment advocated is more heroic than that generally taught, but it has been accepted only after careful consideration of the reasoning of those who hold the opinion contrary to my own. Every case must be individually studied, and the patient's chances of death, of life, with subsequent epilepsy or insanity, or of return to perfect health, carefully weighed; but for a working rule to guide the student and practitioner I think experience will show that the indications given in the table are correct. Trephining, properly performed, is in itself so free from danger that in a doubtful case the patient had better be trephined than allowed to run the risk of death, epilepsy, or insanity. Legouest was very nearly right when he said: "Whenever there is a doubt whether trephining should be done, this doubt is probably an indication that operation should be performed."[1]

[1] Lucas-Championnière, Trépanation guidée per les localisations cérébrales, p. 23.

Syllabus of the Treatment of Fractures of the Cranium.

CLOSED FISSURED FRACTURES.

1. No evident depression, no brain symptoms. No operation.
2. No evident depression, with brain symptoms. Incise scalp and trephine.
3. With evident depression, no brain symptoms. Incise scalp and possibly trephine. (See note below.)
4. With evident depression, with brain symptoms. Incise scalp and trephine.

CLOSED COMMINUTED FRACTURES.

5. No evident depression, no brain symptoms. Incise scalp and probably trephine. (See note below.)
6. No evident depression, with brain symptoms. Incise scalp and trephine.
7. With evident depression, no brain symptoms. Incise scalp and trephine.
8. With evident depression, with brain symptoms. Incise scalp and trephine.

OPEN FISSURED FRACTURES.

9. No evident depression, no brain symptoms. No operation, but treat wound.
10. No evident depression, with brain symptoms. Trephine.
11. With evident depression, no brain symptoms. Possibly trephine. (See note below.)
12. With evident depression, with brain symptoms. Trephine.

OPEN COMMINUTED FRACTURES.

13. No evident depression, no brain symptoms. Probably trephine. (See note below.)
14. No evident depression, with brain symptoms. Trephine.
15. With evident depression, no brain symptoms. Trephine.
16. With evident depression, with brain symptoms. Trephine.

PUNCTURED AND GUNSHOT FRACTURES.

17. In all cases and under all circumstances. Trephine.

Note.—In classes 3 and 11 I should be inclined to trephine if the depression was marked, or the fissures sufficiently multiple to approach the character of a comminuted fracture. In classes 5 and 13 I should trephine, unless the comminution was found to be inconsiderable.

The operation, when decided upon, should be performed at once, or certainly not delayed more than a few hours.

All cases, whether trephined or not, should be treated as cases of incipient inflammation of the brain.

B. In deciding upon operative treatment of intracranial hemorrhage, two questions are to be answered:—

What symptoms render perforation of the skull, in order to remove the clot and secure the bleeding vessels, unjustifiable?

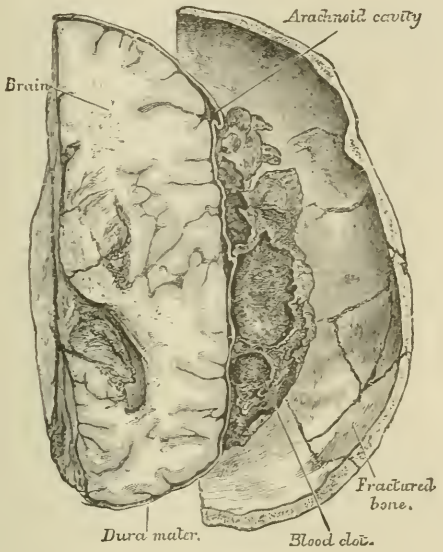
What symptoms demand such operation and render it justifiable?

Intracranial hemorrhages occur between the cranium and dura mater, in the cavity of the arachnoid, in the meshes of the pia mater, in the substance of the brain and in the ventricles. Hemorrhages within the ventricles or in the substance of the brain, whether traumatic or idiopathic, must with our present knowledge be treated solely by medical means; though I consider it not impossible that medical science may in time teach us to locate and diagnosticate such lesions with a precision that may justify exploratory and therapeutic operations in a limited number of cases. Blood poured out in the meshes of the pia mater is usually associated with laceration of the brain, and spreads so widely over the surface of the convolutions, that it is impossible to locate the origin of the bleeding or to remove the distributed blood. Hence, operation is contra-indicated in cases of intracranial bleeding that do not present the symptoms which are believed to be produced by accumulation of blood in either the cavity of the arachnoid or the space between the skull and dura mater.

The differential diagnosis between circumscribed hemorrhage within the arachnoid cavity and circumscribed hemorrhage between the dura and cranium is practically impossible, though it is said that marked inequality of the pupils and marked hemiplegia are less noticeable in arachnoid extravasation than in subcranial extravasation. If localized hemorrhage within the arachnoid is reasonably ascertained, trephining with incision of the dura is in my opinion justifiable. After the bone is removed

the bluish and non-pulsatile dura will probably bulge into the opening, and should be incised to allow escape of the blood. Nancrede refers[1] to cases where the bulging of the dura did not occur until many hours had elapsed. It is not likely that any special vessel will require ligation or similar treatment in arachnoid hemorrhage; as occurs not infrequently in subcranial hemorrhage, where the bleeding often comes from a large meningeal artery or a venous sinus.

Fig. 10.



Hemorrhage from Meningeal Artery. (BRYANT.)

Hemorrhage between the dura and cranium (subcranial extravasation) is not very unusual as an accompaniment of head injuries, because the branches of the large middle meningeal artery ramify over the inner surface of the parietal bone, which, from its position, so often bears the brunt of the vulnerating force. Indeed, the artery is sometimes, especially at the lower anterior angle of the bone, enclosed in a bony canal, so that fracture of the bone at that point almost insures the laceration of

[1] International Encyclopædia of Surgery, vol. v. p. 51.

the artery. The venous sinuses are also torn or wounded at times, and cause subcranial extravasation. Hemiplegia on the side opposite the extravasation, rapidly followed by general paralysis and coma, is frequently seen in subcranial hemorrhage. Convulsions during the time of bleeding occur, though not very frequently.

In bleeding from the middle meningeal artery, the clot often causes pressure at the base of the skull, and therefore may, by pressure on the cavernous sinus and nerves going to the eye, cause on the side of injury protrusion of the ball and dilatation of the pupil, or wider dilatation than in the other eye; while the hemiplegia from cortical pressure will be on the opposite side. If a fissure exists the blood may escape into the temporal fossa of the injured side and cause tumefaction there. Erichsen records two cases where these symptoms were a valuable aid in diagnosis.[1] Such symptoms, as well as embarrassment of respiration, indicate operation; when the blood lies within the brain, because due to cerebral laceration, the patient has, if the laceration is at all severe, no interval of consciousness after the injury; concussion symptoms soon give place to compression symptoms; and paralysis is not complete, as a rule, and is apt to be limited to hemiplegia. Twitching of the limbs, convulsions of the body, restlessness, and muttering incoherence, contracted or irregularly dilated and contracted pupils with perhaps squinting, are the prominent symptoms which occur.

The diagnosis of traumatic subcranial hemorrhage, and indeed, in a surgical sense, the same may be said of arachnoid hemorrhage also, centres upon the absence of the characteristic symptoms of laceration just given, and upon the fact that the group of symptoms commonly called "compression of the brain" occur not immediately after the injury, as in laceration of the brain or hemorrhage into the ventricles or brain substance, but later, that is, after the interval of a few minutes or hours. For example, a man is struck on the head and falls stunned, but soon recovers consciousness, though subsequently he becomes coma-

[1] Surgery, vol. i. p. 735.

tose and paralyzed on the side opposite the injury. Here the presumption of a subcranial or arachnoid hemorrhage having occurred, as soon as reaction has sent enough blood to the head to distend the torn vessel, is very great. This period of immunity from head symptoms after traumatism is sufficient to justify operative search, when an injury or such symptoms as coma, dilated pupil, hemiplegia, monoplegia, or monospasm, sufficiently localize the effusion; or if the symptoms are increasing or are of sufficient severity to point to the probability of active encephalitis. Apoplexy, drunkenness, opium poisoning, uræmic coma and intracranial hemorrhage are often to be differentiated in cases without history. The problem is frequently difficult. Reference has been made, on page 37, to Dr. Seguin's suggestion regarding the diagnosis of latent apoplectic paralysis. A very short interval indicates rapidly flowing hemorrhage and demands early operation, with probably ligation of the middle meningeal artery or one of its large branches, or ligation or compression of a sinus. It must be remembered also that compression symptoms, coming on immediately or quite soon after depressed cranial fracture, are very often due to the subcranial clot rather than the depressed bone. Removal or elevation of bone is often needed more for liberation of blood-clot and control of hemorrhage than for deformity in the bony outline.

Rapid hemorrhage will probably be from the middle meningeal artery, and therefore a large trephine should be put over the anterior inferior angle of the parietal bone, and the squamous portion of the temporal bone just below this angle, unless the localizing symptoms direct to some other point. Inspection of the skull will show that the two large branches of this artery, as they ascend to distribute branches to the lateral region of the cranium, can both be uncovered by a large trephine applied at this point. This point is situated about $1\frac{1}{2}$ inches behind the external angular process of the frontal bone and 1 inch above the zygoma, or $1\frac{1}{2}$ inches directly above the condyle of the jaw.

The rules given by Keen and Erichsen are accurate,[1] I think,

[1] Holden's Medical and Surgical Landmarks. Edited by W. W. Keen, Phila., 1881, p. 18. Erichsen's Science and Art of Surgery, i. p. 739. See Fig. 9, p. 41.

for uncovering the superior branch, but place the trephine a little too high, if the two branches are to be uncovered at their point of divergence. There are probably variations of the point of bifurcation. The bleeding vessel when found may be ligated or twisted, or the bony canal plugged with a piece of stick or wax, or the actual cautery applied by means of a red hot needle. Nancrede[1] has collected 40 cases of operation of this kind with 24 recoveries and 16 deaths. Early operations gave, as might be expected, a much better percentage of recovery than those in which operation was delayed.

The tendency has been to deter surgeons from operating in intracranial extravasations, unless the certainty of diagnosis was great. For example, Gross says:[2] "The only case in which such a procedure is really warrantable, is where the extravasation is associated with, or dependent upon, fracture of the skull, complicated with depression, or serious injury of the soft parts, or where the fracture is situated directly over the course of the middle meningeal artery." This advice is, in my opinion, too restrictive. Gradually increasing brain symptoms after head injury, especially if there has been a period of return to consciousness after shock of traumatism, suggest the occurrence of intracranial bleeding from meningeal vessels, even when no fracture or depression of bone is detectable. Operation is then especially indicated, if the localizing symptoms point to no other region of the cortex and if symptoms such as are described on p. 54 do not render cerebral laceration probable.

C. I come now to the operative treatment of intracranial suppuration.

I believe that operative evacuation should be undertaken in all cases of localized intracranial suppuration, whether the abscess is subcranial, intrameningeal, or cerebral. Ashhurst[3] and some other authorities rather deprecate operation, because patients have lived a long time with cerebral abscess in a quiescent,

[1] International Encyclopædia of Surgery, vol. v. p. 48.
[2] System of Surgery, vol. ii. p. 55, ed. 1882.
[3] Principles and Practice of Surgery, p. 326.

encysted state; and, perhaps, also, because autopsies have in rare instances shown what appear to be abscesses of the brain cured by caseation. The tendency, however, of intracranial suppuration, whether diffused or localized, is known to be towards death. Cerebral œdema, progressive invasion of vital centres, rupture of the abscess into the ventricles, and extension to the surface of the base where fatal meningitis occurs, are the usual modes of destroying life. Spontaneous evacuation through the nares, orbit, ear, or bony wall is too rare to be expected in any given case. Absorption, as far as we know, never occurs.

The difficulty in every case is that of establishing a diagnosis of pus, and proving that the pus, if present, is not a diffused meningeal exudation on the surface of the brain. The occurrence of symptoms indicative of intracranial suppuration, within a few days of the receipt of a cranial injury, usually means diffuse meningeal suppuration and contra-indicates efforts at evacuation. Symptoms of intracranial suppuration occurring at a later period are more apt to be due to abscess in the cerebral tissue, or to a localized pus collection between the bone and the membranes, and therefore indicate more decidedly the propriety of operation with a view to evacuation.

Intracranial suppuration, such as may be benefited by operation, provided that the exact situation of the pus can be diagnosticated, is apt to present a combination of some of the following symptoms:—

1. Headache, or a subjective sense of pressure on the brain. The headache is, perhaps, usually dull, and when present is quite constant.
2. Giddiness, nausea, or even vomiting.
3. Mental hebetude, dulness of senses, slowness in speech and movements, aphasia, mental irritability, muttering delirium, increasing imbecility.
4. Constipation.
5. Paresis, sensory abnormalities.
6. Sleeplessness, optical delusions, attacks of terror.
7. Chills.

8. General convulsive seizures of an epileptiform character; sometimes in a few cases localized convulsions.

9. Little, if any, elevation of general bodily temperature.

10. Perhaps localized increase of surface temperature of the head.[1]

At a later period there will occur most probably—

1. Hemiplegia.

2. Perhaps active delirium, severe headache, high temperature, especially if meninges become involved secondarily.

3. Coma.

4. Passive retinal congestion.

5. Involuntary evacuations.

6. Slow and full pulse, dilated or sluggish pupils.

7. Respiratory failure.

8. Death.

The symptoms of chronic abscess may be so insidious as to escape notice of all except a very careful observer. It has been said that there are at times no symptoms. This I doubt, believing that a critical examination will detect them, especially if the observer be trained in investigating nervous diseases and be sufficiently intimate with the patient to have known his previous habits and mental characteristics.

Puffy swelling of the scalp, and separation of the pericranium, with dry and yellow bone beneath it seen at the bottom of a head wound, will in some few cases be found, and with general symptoms of pus, become a clear evidence of abscess beneath the bone at that spot.

Symptoms, such as just mentioned, demand trephining. If the pus is not found beneath the bone, and the dura is pulseless and bulging, that membrane should be incised. If no pus is discovered, and the general symptoms of intracranial abscess are marked, exploratory puncture and aspiration of the brain should then be performed. In some cases, the surface thermometer may

[1] The suggestion of Flitner to measure the temperature within the external auditory meatuses by a properly constructed thermometer might be utilized. See Journal of American Medical Association, Nov. 15, 1884.

indicate the point on the cranium nearest the focus of suppurative encephalitis.

Nancrede[1] believes that when an intracranial abscess involves cerebral tissue alone the general bodily temperature is subnormal, or at least is not an elevated temperature. He looks upon high temperature as an evidence of localized suppurative arachnitis, or a meningitis in addition to the cerebral abscess. Cerebral abscess secondary to bone disease would, of course, be liable to present an elevation of temperature, because meningitis was an early concomitant.

If a majority of the symptoms tabulated as occurring in intracranial abscess exist and there has been received an injury on the side of the head opposite the hemiplegia, to which the supposed abscess can, with reason, be attributed, an exploratory operation should be done at the point of injury.

If the said symptoms exist and the principles of cerebral localization indicate any special region of the cortex or brain substance, an exploratory operation should be done on that part of the skull that affords best access to the supposed seat of lesion.

Incision of the membranes, and exploratory puncture of the brain substance, with aspirating tubes, to the depth of three or more inches in various directions is justifiable if the symptoms of abscess are undoubted. The abscess cavity or cavities when found should be drained by free openings and counter-openings, and drainage-tubes; and should even be washed out by hydrostatic irrigation, if it is probable that there are pockets in which pus may remain and decompose.

Scarcely any limitation should be dogmatically given regarding the operative search for pus in undoubted abscess of the cerebral tissue, so long as the aspirating tube is not thrust deeply towards the vital centres located in the medulla oblongata and base of the brain. It is never too late to seek for the pus, for Nancrede gave a patient several days of life after he was thought, by several competent observers, to be dead, by promptly opening by incision a cerebral abscess. The earlier the operation is

[1] International Encyclopædia of Surgery, vol. v. p. 86.

performed the better; but it is never too late to make the attempt at evacuating an intracranial abscess.

D. In epilepsy following injury much good may at times be effected by the removal of the cranial wall at the site of traumatism.

An internal spicule, or osteophyte from depressed or chronically inflamed bone, penetrating or pressing upon the membranes, thickened bone, pathological changes in the membranes themselves, the existence of a sequestrum of necrotic bone, depressed and neuralgic cicatrices of the soft parts and similar kinds of irritation, may be the exciting cause giving rise to epilepsy in patients whose convulsive centres are especially irritable. Briggs has seen cases of epilepsy in which narrow spurs of bone nearly a half inch long grew from the inner surface of the skull.[1] Removal of the cause by operation is in such case certainly demanded. Epilepsy due to peripheral irritation of a cicatrix in the scalp without bone lesion may be relieved by dissecting out the scar tissue or detaching it as a flap from the bone and allowing healing by granulation to occur. The application of the actual cautery to the scar may, perhaps, destroy the focus of irritation; though in a case where I tried this method no relief was obtained.[2]

Cauterization or excision of scar tissue should be attempted in traumatic epilepsy therefore before resort is had to trephining, except, perhaps, in cases where there is an evident accentuated depression of the bone. Then the irritating cause of the convulsions is not likely to be in the soft tissues alone.

The subject of trephining in epilepsy was so ably and thoroughly handled at the last meeting of the Association by our present President, Dr. Briggs,[3] that I shall do little more than repeat some of his statements and add a few thoughts which may serve to impress the Fellows with the importance of the topic and cause them to again read his valuable paper.

[1] See Duret's paper on the Dura Mater, to which reference is made on p. 43.

[2] See TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, 1884, p. 132.

[3] Ibid.

The development of epileptic convulsions requires, first, a persistent abnormal excitability of the centres in the medulla oblongata and pons Varolii; and, secondly, a peripheral irritant. The latter is the exciting cause, and may be hard to discover in any given case. Traumatism often furnishes the peripheral irritant in the existence of a sensitive scar in some part of the head or body, or a cranial lesion irritating the cerebral membranes or brain. The hyper-excitability of the medulla and pons may be inherited, or acquired through personal accidental causes. Echeverria attributes[1] ten per cent. of all epilepsies to injuries of the head; but it must be remembered that the convulsions may not be developed for years after the injury. Briggs has seen cases in which ten years had elapsed, and he quotes a case of Dudley's which developed sixteen years after injury, and was entirely cured by trephining. I must admit, however, that Althaus has stated that fourteen years' experience in a hospital devoted to epilepsy has taught him that traumatic epilepsy is rare.[2]

If operative treatment is instituted for traumatic epilepsy, all the diseased or depressed bone should be removed. Briggs has taken away the cranial wall covering an area as large as the palm of the hand, and cured the epilepsy without causing an unpleasant symptom.

From tables of the operation collated by Stephen Smith, Billings, and Echeverria, 92 American operations are reported, of which 63 were cured, 13 ameliorated, 2 not changed, 14 died.[3] Walsham has collected 82 cases, to which he has added 48 collected by Billings and others, making 130 in all, of which number 75 were completely cured, 18 improved, 7 unimproved or worse, 30 died.[4] 30 cases of epilepsy from old injuries of the head have been operated upon by Briggs. Of these, 25 were cured, 3 ameliorated, 1 not changed, 1 died.

In 48 of 82 cases, collected by Walsham[5] himself, cure was

[1] Briggs, see TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, 1884.
[2] Amer. Jour. Med. Sciences, January, 1880, p. 270.
[3] Quoted from Briggs.
[4] St. Bartholomew's Hosp. Reports, 1883, p. 130.
[5] Ibid., pp. 130 and 136.

effected, 13 were relieved at the time of report, 4 were not improved, and 17 died; which gives a death-ratio of 20.7+ per cent., but some of the deaths occurred so long after the operation as to be scarcely attributed with justice to the operation.

Nancrede says[1] that after adding 37 cases collected by himself to the whole number collected by Walsham (130), he finds in the 167 cases a mortality of 19.16 per cent., but does not describe the kind of cases in which death occurred. Cure has in a number of cases followed operation, though no pathological change was recognized in bone or membranes.

In epilepsy and insanity from traumatic causes, connected with the skull, the lesions appear, according to Walsham, to have generally existed in the anterior half segment of the cranium.[2]

The symptoms justifying operation are cicatrices which are sensitive, tender, or abnormally hot by the surface thermometer; pain or sense of brain pressure at the seat of old injury, which may not be constant but which is always, when present, referred to the same spot; pain, vertigo, or convulsions produced by pressure upon the cicatrix; depressions or elevations in the bone; fistules leading to necrotic bone; and any symptoms indicating through study of cerebral localization brain lesions in the region of the external scar. Operation seems to me to be especially indicated in these cases if the scar is in front of the middle of the cranium.

The symptoms contra-indicating operation on the head are the existence of other foci of irritation, as in one of Briggs's patients who had a depression of the skull and diseased bone in one leg, and in whom removal of the necrotic tissue in the leg cured the epilepsy without any surgical interference with the head. The absence of pain, tenderness, or other symptom of irritation, the history of epilepsy in other members of the family, or a short period of medicinal treatment would be facts that should cause hesitation in operation until every other means has been

[1] International Encyclopædia of Surgery, vol. v. p. 101.

[2] On Trephining the Skull in Traumatic Epilepsy, St. Bartholomew's Hospital Reports, 1883, p. 139.

employed, or until the progressive character or the severity of the epilepsy forebodes early impairment of intellectual functions.

If operation fails to relieve, it may be repeated near the old spot of operation after the lapse of several months; for at least one case is known where operation was ineffectual, and subsequent autopsy showed a fragment of bone pressing upon the encephalon. Removal of this would probably have cured the epilepsy. Several months' time must elapse before the operation is declared unsuccessful; for the brain does not always give up its convulsive habit at once. Subsequent treatment to relieve the hyper-excitability of the medulla and pons and to remove any other causes of peripheral irritation must be judiciously carried out. The cicatrix left after trephining may become a secondary peripheral irritant and need excision to relieve pinched nerve filaments. I know, however, of no such case on record.

How long should operation be delayed in epilepsy presumably caused by a depressed fracture of the skull or other localized traumatic irritation? Certainly not at all after the diagnosis of the cause has been made, provided that medical means have been efficiently tried and have failed. Operations done promptly prevent an increased development of abnormal excitability in the convulsive centres, and give, therefore, the best chance of recovery from the epileptic manifestations. Mental deterioration increases with the lapse of time, as does, in my opinion, the risk of serious cerebral reaction after operation. A normal nervous system would be less likely to assume inflammatory symptoms dangerous to life than would a nervous tissue long subjected to irritating influences. If the epilepsy cannot be definitely attributed to the traumatism which by the history and by the scar is known to have occurred, I should advocate an empirical operation in the course of five or six months or even earlier; provided that no other cause is discovered, that medicinal treatment has proved unavailing, and that the epilepsy is increasing in severity.

E. Operations for the relief of insanity due to cranial traumatism have not been very generally advocated or attempted. One

year ago, Dr. William A. Byrd brought the subject to the attention of this Association,[1] and referred to four cases which were trephined for insanity after fractured skull. Three were operated upon by Dr. W. T. Briggs; the fourth by Dr. Byrd himself. Dr. Byrd's case improved for a short time and then became progressively worse in regard to her mental condition. Two of Dr. Briggs's cases entirely recovered their mental health; one was a boy; the other a man, who had received a gunshot fracture above the ear. The third of Briggs's cases was an almost hopeless one, which he had at one time declined to subject to operation. The lamentable condition of the imbecile patient and the solicitations of the family finally induced him to operate. Death occurred on the second day, after "a rigor followed by great depression."

In the discussion following Dr. Byrd's paper, Dr. T. F. Prewitt is reported[2] as having spoken of trephining an insane woman, who was relieved only for a time. The report here is incorrect, for Dr. Prewitt has written me that he has never trephined for insanity. The remarks, I think, should be ascribed to Dr. Byrd, and refer to his case which formed the subject of debate.

I find a case of insanity cured apparently by operation, mentioned by Dr. L. A. Stimson, and another by Dr. J. L. Little.[3] In his elaborate table of trephining cases, Amidon[4] refers to McCormick's case of acute mania, occurring eight years after a depressed cranial fracture, which recovered after the depressed area of bone was removed by operation. In this instance there was pain at the site of wound, which was situated at the junction of the sagittal and coronal sutures.

With so few cases as are reported it is difficult to formulate dogmatic rules. It seems to me, however, that the operation of removing a portion of the cranium is justifiable when, within a

[1] Transactions of the American Surgical Association, Vol. II., 1884, p. 130.

[2] Ibid., p. 134.

[3] Annals of Anatomy and Surgery, 1883, viii. p. 137.

[4] Annals of Surgery, March, 1885, p. 211, quoted from American Practitioner, 1883, xxvii. p. 237.

few months after a depressed fracture of the skull, progressive mental aberration occurs in a previously sane patient who presents no other assignable cause for the intellectual malady. If there is pain or tenderness at the site of the injury, or if motor or sensory phenomena are found in the regions supplied by the nervous centres situated under the seat of injury, the demand for operation is much more imperative. I admit that cases reported as cured of acute maniacal symptoms by trephining are doubtful cures, because they might have recovered without operation. A number of chronic cases recovering after operation would afford much more conclusive evidence of the value of operative therapeutics.

Operation should be done before the pathological changes cause great imbecility. If pressure causes local degeneration or atrophy of the cerebral convolutions, it is imperative to remove that pressure as soon as it is evident that remedial drugs afford no relief and that the mental impairment is progressive. In an early part of this paper I have referred to a case seen by Dr. Mills and myself, in which I was prepared to trephine as soon as it was clearly established that the mental deterioration had no other assignable cause than the depressed fracture. Unfortunately the patient passed from observation.

It is impossible to fix any time; but progressive failure during three months would incline one to operate, and the same condition during six months would, I think, justify the surgeon in very strongly urging surgical treatment. On this point, however, I should like to hear the views of the Fellows of the Association.

In operating, all the depressed bone should be removed by cutting away the intervening bone between the holes made by numerous applications of the trephine. The Hey's saw, gnawing forceps, or burr attached to the surgical engine will accomplish this. Guthrie[1] refers to a case of fracture, but not of insanity, in which twelve disks were removed before the depressed bone was properly elevated. Recovery followed.

[1] Gross's System of Surgery, vol. ii. p. 88.

Operation is not justifiable when there is evidence that some degree of insanity existed before the injury, nor when the patient has had, previous to the injury, insane delusions, not traumatic, from which he had recovered before the traumatism; nor when the character of the insanity is such that the pathological change is probably located in a region of the brain, distant from the seat of injury; nor when the insanity involves so many intellectual functions that general cerebral disease is clearly existent.

F. Operations for the removal of cerebral tumors have, as far as I know, been attempted only recently. The fear of approaching the brain with instruments deterred surgeons in a manner that seems unaccountable, when we recall the numerous instances of recovery from accidental brain injuries, that have been reported during the last fifty years. It is probable that most of those which recovered were of a character that insured efficient drainage, and that we did not have discernment enough to realize the true method by which fatal results were averted.

Primary cerebral tumors, which are not controlled by medicinal treatment, are usually single, seldom give rise to secondary deposits, are generally surrounded by an inflammatory zone of demarcation, and continue to increase in size until they destroy life. Therefore, when they are believed to be upon or in the cortex of the brain, or at least not deeply located in the cerebral substance, they should be removed by operation, unless they are in some such inaccessible and especially dangerous region as at the base of the brain.

Amidon[1] was one of the first, if not the first, to insist upon this method of dealing with cerebral tumors. A few months ago, Godlee removed by enucleation a glioma the size of a walnut, situated a quarter of an inch below the surface of the ascending frontal convolution, and which had been located by Bennet.[2] For 20 days the patient did well; but he died on the 28th day

[1] Med. News, June 21, 1884.

[2] Lancet, Dec. 20, 1884, and Jan. 3, 1885. See Amidon's discussion of its details in Annals of Surgery, March, 1885.

from meningitis at the lower part of the wound, spreading down towards the base of the brain, on the same side, the whole of which was inflamed and covered with plastic lymph. The rest of the brain was normal. Amidon justly asks whether death might not have been due to the absence of antiseptic precautions, and the want of provision for drainage, especially as the galvano-cautery was used to stop bleeding. I am not aware that the operator has definitely stated whether antiseptics were adopted and provision for drainage made.

This remarkable case of sagacious diagnosis by cerebral localization followed by a well-planned surgical operation is certainly very encouraging, and will doubtless be the incentive which will save many lives.

Multiple cerebral tumors, tumors of the base, and cerebral growths, secondary to neoplasm in other localities, should not be subjected to operation. If other circumstances obtain, and the symptoms are such as will localize the growth with reasonable certainty, excision should be attempted.

Sands records[1] a remarkable case in which Dr. Amidon diagnosed traumatic epilepsy in a patient who had struck the left parietal region against a sharp corner, but in which trephining showed no bone lesion. Death occurred in eight days, and a gummy tumor which could probably have been removed was found directly beneath the seat of trephining. No evidence of meningitis from the operation was apparent. This case may have been a potent factor in determining Dr. Amidon's well-known advocacy of the enucleation of cerebral tumors.

Dr. Charles K. Mills, who has made at least 25 to 30 autopsies in cases of brain tumor, of which number twelve have been published,[2] has called my attention to the fact that in several of them removal of the tumor would have been readily possible.

I give from his paper mentioned an abstract of two cases:—

Case IV.—Clinical history: Female, æt. 30. No history of causation. Headache continuous, sometimes agonizing. Percussion of

[1] Annals of Anatomy and Surgery, 1883, viii. p. 113.

[2] Archives of Medicine, Aug. 1882.

head caused most pain in right parietal region. Vomiting when headache was most severe. Vertigo. Mind clear, but acted slowly; emotional. Spasms, beginning with twitching of fingers of left hand; most severe on left side, and especially in left arm. Upper as well as lower fibres of left facial nerve partially paralyzed; nearly complete paralysis of left arm; slight paralysis of left leg. Bowels and bladder partially paralyzed. Impaired sensibility of limbs of left side. Left patellar reflex diminished. Sight very imperfect. Choked disks. Hearing defective in right ear.

Pathological anatomy: Carcinoma, about one-and-a-half inch in diameter beneath and adherent to the pia mater of the convexity of the right hemisphere; the pia and dura mater were united by strong adhesions. The tumor involved the middle portion of the ascending parietal convolution and the upper part of the inferior parietal lobule, pushing aside the inter-parietal fissure. The anterior extremity of the tumor was about one-fifth of an inch back of the centre of the fissure of Rolando. On the inner side of the tumor the white matter of the brain was broken down.

Case V.—Clinical history: Female, æt. 38. History of syphilis. Blows on the head. Headache with agonizing paroxysms. Top and right side of head sensitive to percussion; and headaches severest in these regions. Vomiting. Vertigo. Great mental irritability. Severe left-sided spasm, beginning with twitching in left toes and foot. Partial paralysis of right leg and arm, most marked in leg. Hyperæsthesia. Impaired sight. Choked disk. Head temperature taken once; right parietal region, 97.2° F.; left parietal region, 96° F.

Pathological anatomy: Gumma. Attached to the fused membranes of the right convexity. Involved the upper one-fourth of ascending frontal, and a smaller segment of the ascending parietal convolution, crossing the fissure of Rolando at its upper extremity. A good example of strictly cortical lesion, involving only gray matter, and having only a very thin layer of softened tissue on its inner side. Microscopic examination of the optic nerves showed the appearance of choked disk, with probable ascending neuritis.

I have thus considered most of the topics which may be included under the head of cerebral surgery. In fractures, hemorrhage, and abscess, perforation of the skull will soon become

quite common. In epilepsy, insanity, and tumor the profession will be more tardy in opening the skull. While I am sure that these conditions, in certain instances, can only be treated successfully by operative procedures, I believe also that other cerebral conditions, less well defined in pathology, such, for example, as incurable and intolerable headaches after injury, may in time be justifiably subjected to similar operations.

For such operations upon the skull and brain proper instruments should be at hand. For uncovering the centres of motion in searching for suspected lesions localized by symptoms, a trephine removing a disk three centimetres in diameter is none too large, for then both ascending convolutions and the intervening fissure of Rolando can be inspected with ease. A trephine with a cutting edge along only a half or two-thirds of its circumference, would, I think, be convenient at times for deepening a groove on one side of the disk, when the remaining portion of the circular groove has already completely cut through the bone in a thinner spot; and also for enlarging a trephine hole previously made. Dr. W. B. Hopkins has suggested a modification of the trephine which may be of value in this connection.[1] A probe with a large olive-shaped head and a light aluminium shaft, as recommended by Fluhrer, is certainly the proper exploring instrument in gunshot wounds of the brain. The head should, as he suggests, be placed so that the probe will be vertical and drop into the wound by its own weight. Aspirating needles should probably be made with a round end and a lateral opening like a catheter.

DISCUSSION.

Dr. Hunter McGuire, of Richmond, Virginia.

I am sorry to say that I am not entitled to speak. I believe that one of the rules is, that those intending to discuss the paper should

[1] Annals of Surgery, July, 1885.

make some preparation for it. Circumstances have prevented me from writing anything out. However, if the Society will pardon me, I will say a few words in regard to the interesting paper of Dr. Roberts. I think that we are indebted to him for the complete and exhaustive article that he has written on the subject. The last part of the paper has been especially interesting to me. This branch of surgery is still in its infancy, and no man can foresee what the future has in store for injuries and diseases of the brain.

In looking over the propositions submitted for discussion, however, I find that there are only two conditions named in that long list where we shall not operate, first, in simple fractures where there is no evident depression and no brain symptoms; second, in open fissured fractures, where there is no evident depression and no brain symptoms. In every other condition, we are distinctly told to trephine, to probably trephine, and occasionally to possibly trephine. The only condition where we do not trephine is where there is no depression and no brain symptoms.

This takes us back about a hundred years, when patients went about bragging of the number of holes they had in their skulls, and doctors bragged of the number of holes they could make and not kill the patient. I think that this is the most dangerous doctrine that we can send out to the world, that we are to trephine for all these injuries of the skull and brain. The truth is that no set rules can be laid down to apply to all cases. Every case stands by itself. We do not find two injuries of the skull alike any more than we find two faces alike.

Taking up the first proposition, it is stated that "the complexus of symptoms called 'compression of the brain' is not due so much to displacing pressure exerted on the brain-substance, as it is to some form or degree of intracranial inflammation."

How does Dr. Roberts account for the almost instantaneous appearance of symptoms of compression of the brain? A man is struck on the head, the bone is fractured and depressed, and in an instant there are symptoms of compression, unconsciousness, slow, labored, and possibly stertorous respiration. These symptoms may appear in five or ten minutes, or they may appear in a few seconds. Is it possible that intracranial inflammation with inflammatory exudation can have taken place so soon? It seems almost impossible to me that inflammation can produce symptoms of compression in so short a time.

Another thing. It is said that "the removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone." It was Cooper or Hunter who said that between the trephine of the surgeon and eternity there was a little thin sheet of paper. How the Doctor can come to the conclusion that trephining is so simple an operation is beyond my comprehension. I do not like to joke about a serious subject, but the author stated that a man might be trephined and go to his home, but he forgot to tell us which of his homes, whether the long home or the one here.

Again. "Drainage is more essential in wounds of the brain than in wounds of other structures."

I believe that the statement is correct. I think that there is no condition in which drainage is more essential than in these injuries. In the last case I trephined, instead of making the semilunar flap, which is an improvement on the old crucial incision, I took out a section of the scalp just the size of the trephine in order that drainage might be perfect.

"In the majority of cranial fractures the inner table is more extensively shattered and splintered than the outer table."

I have a good illustration of that in my pocket. This button of bone I took from a man who had epilepsy from a punctured fracture of the skull. This had been going on for two or three years, and the man's mind was affected. I could find nothing but the mark in the scalp to show the seat of injury. There was no depression of the bone, and even after the incision of the scalp was made, no evidence of depression could be detected. Upon the inner surface of this button you will see a sharp spiculum of bone. The dura mater and arachnoid were attached to this plate, and in separating it, in consequence of the great intracranial pressure, the cerebro-spinal fluid almost spurted out. The man recovered from the operation, but, as the case is a recent one, the effect on the epilepsy is still uncertain.

If you will pardon me, I shall relate the case of a negro man in the Central Lunatic Asylum of Richmond. He had been left there by the Freedman's Bureau. We had no history of the case, and none could be obtained. He would sit for hours crouching by the wall of his room, and no one in the hospital had ever heard him speak. He had frequent epileptic fits and occasional attacks of

violent mania. I trephined his skull and removed the depressed bone—a part of the left parietal bone. A few hours later, I went into the room and said "Good morning." He replied "Good morning; where is the army to-day?" That was the first intelligent word he had uttered. I said, "Where was the army yesterday?" "It was at Manassas," he replied, referring to a period about nine years previous. Between this period and the time of operation, nine years, his mind was a perfect blank. He told me afterwards, that he had been struck with a boat hook by a companion. He remembered the man approaching him with the hook, and then lost all recollection until the removal of the bone. This man recovered, and worked as a common laborer for years after.

Dr. Moses Gunn, of Chicago, Illinois.

I have very little to say on this subject from the fact that I am on record. Three years ago I covered some of the ground gone over to-day. At that time I took occasion to advocate a more frequent use of the trephine, and I undertook to establish the truth of the position, that in all cases of fracture of the cranial walls with depression, where the injury was a simple one, or a closed one as Dr. Roberts has it, the indication should be to elevate it, as it had been the universal rule in compound fracture, and I held that the advances made by reason of antiseptic and aseptic procedures, gave us such advantages as would enable us with impunity to convert a closed or simple cranial fracture into a compound one. In discussing this subject before my classes, I have been in the habit of stating certain propositions to them, and in one instance I have made it one of the questions in the final examination. Given two cases of fracture of the skull with depression in persons in whom there is a parity of physical condition, a parity of age, a parity of vital force to resist injury and to repair injury; in the one instance that depression shall be associated with laceration of the tissues covering it, while in the other the fracture is closed; what is the treatment in each instance? I have no hesitation in saying that, under such circumstances, a simple fracture should be converted into a compound fracture, and that the mere division of the skin, opening-up of the fracture to the atmosphere, is not attended with any increase of danger under present circumstances. I took that ground three years ago. In response to a request from our greatly venerated and now lamented President, Dr. Gross, who

asked me to prepare an article upon the treatment of fractures of the skull, acute and chronic, I wrote the paper in which I took this ground and advocated the doctrine which I have now indicated. I remember that I was sharply criticized. My friend from Richmond used almost the same words that he has done to-day, saying that it was dangerous doctrine, that it was a warrant to young men to resort to the indiscriminate use of the trephine. I remember that my friend from South Carolina, Dr. Kinloch, took a similar ground. They criticized me severely. The *London Lancet*, in noticing the Proceedings of the Association, said that it was unsound doctrine. Therefore, I say that I am on record, and have little to say. I am glad to say that I had the indorsement of our present worthy President when he spoke on this subject two years later. I am delighted to-day with the paper of my young friend from Philadelphia, and I propose to criticize his doctrine very slightly.

In regard to the use of the trephine, the mere act of the removal of a portion of the cranial walls is not a dangerous operation. In fact, three years ago I said "this is not an operation fraught with danger;" the expression, "fraught with danger," being quoted from a work on surgery. I believe that it is simple and harmless, and that pretty extensive removal of the cranial wall can be made with the trephine with impunity. Therefore I am prepared to indorse all that he has said, and to say that the rules which he has given, if applied with good sound judgment, can be applied not simply with safety, but with benefit to the human race. This is all that I have to say on that point.

There is another point upon which Dr. McGuire has touched, and I coincide with him. That is in regard to this proposition, "The complexus of symptoms called 'compression of the brain' is not due so much to displacing pressure exerted on the brain-substance as it is to some form or degree of intracranial inflammation." That this is true in a great number of cases, I believe. I believe that in many instances where compression of the brain, so called, is spoken of, that it is not simple compression, but the result of irritation and inflammation. As far as compression of the brain in itself is concerned, it is not an important symptom. As Dr. Briggs said three years ago, "If compression of the brain were the only effect produced by a depressed fracture, then I should never use the trephine, for in a short time the brain would accommodate

itself to the mechanical compression." That is true. The danger is in the irritation and inflammation and changes in nutrition which follow, resulting, perhaps, in augmentation of bone-tissue, leading to the formation of osteophytes from the new bone-tissue thrown out in the process of healing. It is from these sources that the danger comes, not merely from the compression.

But I am not willing to say that there is no such thing as mechanical compression. As Dr. McGuire has said, a man may receive a depressed fracture from a blow not sufficient to produce concussion, and is in an instant picked up with symptoms of compression. He has a slow, full pulse, a warm surface, and a good capillary circulation, but he is in a state of profound coma, he breathes stertorously, and the alæ of the nose elevate with the movements of respiration. There is no chance for inflammation there. There is mechanical irritation at once; but, as Dr. Briggs has said, if that was all, I should not trephine, as I should be sure that in a short time these would be overcome. The danger is in the fact that we may have irritation and inflammation, an irritation which will become permanent, giving rise to secondary symptoms.

In regard to the freedom of danger from the trephine, I am prepared to say, that, if used with good sound judgment, the position of Dr. Roberts is correct.

Dr. Charles B. Nancrede, of Philadelphia, Pennsylvania.

There are many points in which I entirely agree with the writer of the paper, but there are a number of others in which I cannot agree or go quite so far as he has done.

As to the first proposition, I certainly agree with the previous speakers. If he had called it secondary compression I would agree with him that it was due to some form of intracranial inflammation. I think that it is hardly now worth while to enter upon a discussion as to the true nature of those rare cases in which the symptoms of compression appear immediately, without any injury of the brain substance. The experiments of Fisher, of Berlin, may help to explain them. He has found that irritation of a sensory nerve of the scalp, or of some distant part, produces a spasmodic contraction of the vessels of the brain and temporary abolition of function of the encephalon.

I do not believe, either, that any one who will look at the subject in a proper way will doubt that the conversion of a simple cranial

fracture into a compound one, even with the modern methods of treatment, does add to the danger. The idea of compound fractures is associated more or less unconsciously with compound fractures of the limbs. There is no question that, even with the best methods of treatment, if we convert a simple fracture of the thigh into a compound fracture, we do somewhat endanger life; but to compare such a procedure to a similar one converting a simple cranial fracture into a compound one is so manifestly unfair as to need no discussion; the dangers of the two are not comparable, since they are due to different causes.

"The removal of portions of the cranium by the trephine or other cutting instruments, is, if properly done, attended with little more risk to life than amputation of a finger through the metacarpal bone." Provided no accident happened, that might be absolutely true; but, in considering any operation, you must take into account the complications that may arise. I hardly think that any serious accident could happen during an amputation through a metacarpal bone. In the operation of trephining, I can conceive of damage to the membranes or other accident inherent to the operation which may happen to the most careful operator.

No one doubts the truth of the fourth proposition.

As to the fifth proposition, I cannot think that the cranium should be perforated as an exploratory measure as often as trephining is demanded for therapeutic reasons.

No one questions the importance of drainage.

I must dissent from proposition seventh, that "many regions of the cerebral hemispheres of man may be incised and excised with comparative impunity." I think that there is a decided amount of danger. It has not been done frequently enough to enable us to speak positively. Of the few cases in which it has been done, very many have terminated fatally.

The treatment of wounds of the large veins and arteries which supply the membranes is the next point. Dr. Roberts refers to the case of Dr. Hopkins. There is one point to which I desire to call attention. I think that it is a good rule, when you have reason to suspect that one of the sinuses may be wounded, to act as though it were wounded, for I am cognizant of two cases in good hands in which the patients perished before the surgeons' eyes, because this was not done. In this case of Dr. Hopkins there was a fracture of the upper portion of the parietal bone produced by being thrown

from a train. Dr. Hopkins, without thus planning the operation, was led by the position of the fragments to remove them so as to give ready access to the median line of the cranium. When the last fragment of bone, which lay over the longitudinal sinus, was lifted, there was an appalling gush of blood. A finger on the bleeding point arrested the hemorrhage with much less pressure than would be necessary in bleeding from a vein at the bend of the elbow. We first attempted to tie it laterally, but as soon as the tenaculum or forceps was removed hemorrhage would recur. The repeated attempt at tying resulted in enlargement of the opening to two or three times its original extent. Finally, Dr. Hopkins placed a small square of lint over the bleeding point, and over this other pieces sprinkled with iodoform. The scalp was laid down and some pressure was made by the turns of the bandage used to retain the dressings. There was no further trouble, and the patient is well to-day, six months after the operation.

I have paid considerable attention to cerebral localization. All that I am prepared to venture is, that we are fairly well acquainted with the motor centres, and that by a very careful examination in any given case, we may, with a fair prospect of success, discover and locate the seat of lesion in the motor centres, as the Doctor has shown in his first diagram, which I think is nearly identical with the one which I have given in my article in the *International Encyclopædia of Surgery*. The greatest caution should be exercised in trephining for irritation of the sensory centres. I think that they are more deeply situated than the motor centres. There are few cases of irritation of the brain which have such an isolated lesion that we can be sure what portion of the brain is involved. In irritation of the oculo-motor nerve, how are we to tell that the lesion is not at the base of the brain, for experiment has shown that irritation or destruction of the quadrigeminal bodies will produce the same symptoms as irritation or destruction of the nerve? How can we tell that it is not deep seated, rather than in the centres described?

Now as to crural monospasm. I saw last Saturday, in consultation with Dr. G. A. Rex, a case in which, if there had been any head injury, as there might readily have been, we might have been misled into attributing the symptoms to a brain lesion. The diagnosis reached was thrombosis of the veins or arteries of the anterior tibial nerve, with secondary hemorrhagic neuritis. That had to be

diagnosed from limited anterior polio-myelitis. In such a case, if the patient had, when she fell, cut or even struck her head, we might have trephined for an injury of the cranial centre when the lesion was in the nerve.

I have been in the habit of dividing fractures of the skull into three classes; one in which there was so much concomitant injury of the brain, that whether you trephine or not the patient will die. Although by the elevation of broken fragments you may prevent secondary irritation, yet your operation will have no influence one way or the other, so far as the patient's life is concerned, because the injury is so severe that he must die. These cases are constantly included in trephining statistics, although they should not be.

There is another class at the other extreme, in which the intracranial lesion is so slight, that if secondary trouble can be prevented death will not occur. The secondary trouble is produced by the depressed fracture, and by the pulsatory movements of the brain, driving the dura mater against the fragments, producing fatal encephalitis. If you trephine such a case, you remove *the* cause of death. The intracranial lesion is so slight, that if you can prevent secondary inflammation the patient gets well.

Between these two classes there is a third class of doubtful cases of severe injury which are not necessarily fatal, but are rendered so by the presence of broken fragments. There trephining is indicated. Patients do get well without this operation, but how many after the lapse of ten years are well? Very few.

I do not want to occupy the time of the Association, but there are one or two other points. One is as to the danger of the operation *per se*. I cannot see where Dr. Amidon obtained the statistics giving the mortality as only 3.6 per cent. I have carefully gone over the literature of the subject, and I find the mortality to be 10.69 per cent.

I would not incise the scalp with no evident depression and no brain symptoms, nor would I trephine it in a closed comminuted fracture with no evident depression with brain symptoms. I think I have pretty clearly indicated my position as to where I would trephine and where I would not, and why I would trephine and why I would not.

Now as to the large trephine, I hardly think it necessary, and, as Dr. Roberts has pointed out, it is attended with a certain amount of danger to the membranes.

The Doctor made one statement which may be misleading, and that is in reference to the puffy tumor of Pott as indicating intracranial suppuration. It is an indication of osteomyelitis, and here we frequently have suppuration between the dura mater and the bone. Often, however, no intracranial suppuration is found on trephining such cases.

He also referred to subnormal temperature in cerebral suppuration. If this were confirmed, it would be a valuable sign. As far as I am aware, in all cases of pure cerebral abscess, where the case has been reported with the temperature, there has been normal or subnormal temperature. Even where suppuration supervened on chronic ear disease, the temperature fell to normal or subnormal at the time of the formation of pus.

I would always trephine in a simple, markedly depressed comminuted fracture, when the margins of the cranial opening are irregular and jagged, as far as can be made out. If the depression were slight I should not operate, but would carefully watch, and at the earliest indication—I will not say of intracranial inflammation, for trephining cannot remove inflammation, and, therefore, after inflammation has ensued, there is no use in trephining unless there is pus—but, on the first symptoms of intracranial irritation, I should trephine. These symptoms of irritation can be detected probably long before there is anything more than accumulation of serum.

There is one interesting reference to foreign bodies. When the foreign body has been left, although the patient has recovered, recovery has usually been followed by sequelæ. Where the foreign body has been removed, there has been a larger percentage of recoveries, and recovery has been effected with a comparative immunity of the mental and physical functions.

Dr. Washington F. Peck, of Davenport, Iowa.

It was not my intention to participate in the discussion. My experience leads me to coincide with the majority of the opinions advanced in this paper. I have always believed that the first thing to be done in connection with injuries of the skull is to determine whether or not there is a solution of continuity in the skull, and, if so, whether or not there was pressure on the intracranial contents. I do believe that pressure by bone upon the meninges and cerebral tissue is capable of developing cerebral symptoms directly, and, secondly, we have pressure from the products of inflammation

which undoubtedly call for other interference than therapeutic measures. I have several cases which I shall presently relate which illustrate an experience upon which my opinions are largely based.

Some years ago I saw a brakeman who had been struck with a railroad tie in the temporo-parietal region of the right side. He suffered immediately from symptoms of compression, and with these there was free hemorrhage from laceration of a temporal vessel. He remained in this condition twenty-four hours. I made a trephining operation, boring two holes in the parietal bone, and removed about two inches square of bone. He gradually recovered from the condition of compression, and in about three or four days suffered from a convulsion. I then made a free incision into the brain and evacuated an ounce or an ounce and a half of pus. From that time the recovery was uninterrupted.

In a second case a boy was shot a little left of the centre of the frontal bone. There were immediate symptoms of compression. I saw him within a few hours, and trephined the skull; and through the opening made by the trephine a clot of blood half an inch in diameter escaped. The boy recovered, and is now perfectly well with the bullet in his brain. Had no operation been performed, it is reasonable to presume that the pathological changes of the clot would have furnished conditions favoring septicæmia, and the pressure would have caused convulsions and ultimate death.

Another case is that of an orphan in an orphan institution. The child was struck on the head with a piece of coal, producing a depressed fracture, the depression extending half an inch below the surface. The child was placed in bed, kept quiet, and no unpleasant symptoms have succeeded. Whether the case will ultimately develop epilepsy or insanity, I cannot say. There were no symptoms of compression, and I saw no necessity for operating.

I have had in all about six cases, and it has been my custom to look forward to removing pressure and establishing drainage. I believe with Dr. Gunn, that if pressure is unassociated with any pathological change, except that connected with the process of repair, the operation is not necessary.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

The propositions which the author of the paper has laid down are so sweeping, that the paper should not go out without a thorough

discussion, and the expression of clearly and well-defined views as to the conditions which demand trephining, and the conditions which do not demand trephining.

The first proposition has been sufficiently discussed. We certainly have compression of the brain from other causes than inflammation. It is rare to have compression from depressed bone, but it is not rare to have such symptoms from the presence of blood. I do not think that the author meant to ignore these facts in his proposition.

"The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp is, with the improved methods of treating wounds, attended with very little increased risk to life."

That might be true, and yet it might not be advisable to trephine in every case. It seems to me that it should not be done unless there is good reason. In the first place the injury that results from a blow upon the head is not simply the fracture of the bone, nor is it due mostly to the fact that the bone is depressed; the result is largely due to the injury of the brain-structure and of the meninges. It is not simply that the bone is broken. The fracture of the bone is of no greater importance than a fracture any where else. If left alone, nature will certainly repair the injury so far as the fracture is concerned. The danger is mostly in proportion to the injury of the brain-substance. In reference to that point, the author states that in simple fissured fracture, with no evident depression and no brain symptoms, he would not trephine.

Again he says, "No evident depression with brain symptoms. Incise scalp and trephine." What does he mean by brain symptoms? Does he mean immediate brain symptoms? Are they due to depression or to concussion? To what brain symptoms does he refer? It is a little indefinite.

Again, "With evident depression, no brain symptoms. Incise scalp and possibly trephine." This is pretty sweeping. There might be slight depression and no brain symptoms. There would be slight indications of concussion, and you convert by such an operation a simple fracture into a compound one. It is true that with antiseptic methods we certainly do control inflammation to a certain extent, but you recollect that we do not agree in regard to the value of antiseptics. Many high authorities do not regard antiseptics as of value. I do not mean to say that I do not. While

individually I look upon antiseptics as most valuable in enabling us to control inflammation in most cases, I do not think that it will be claimed by the most sanguine that we can always control inflammation by antiseptics.

Dr. Nancrede has said that a parallelism has been drawn between fractures of the leg and fractures of the skull, and that this is not just. Why is it not? In fracture of the leg, if you expose the parts by a wound, the dangers of inflammation are much greater. You may have a serious fracture which if not opened will go along well enough; you convert it into a compound fracture, and with all your efforts you have no guarantee that you will not have done great injury. I do not see why that shall not apply to the skull as well. We certainly do add to the danger of inflammation when we convert a simple into a compound fracture. The danger is not as great as formerly, and in most cases it is perhaps harmless.

In the case he reports as having trephined, there was a certain amount of depression, but I think that that would have given rise to no particular damage. The bone was smooth, not spiculated, and but little rougher than the normal bone, and it will be seen that he did not raise the depressed bone. If there were no concomitant injury of the brain, I think that a fracture of that kind would give rise to no irritation, and I doubt whether it would have been followed by epileptic attacks. The brain accommodates itself to a certain amount of pressure. The case that Dr. Peck has cited, where a child had a depressed fracture and recovered, is a case in point. It is stated that frequently in children depressed fractures may disappear entirely, and no symptoms follow. It is where the bone is depressed in such a way as to act as an irritant to the membranes and the brain that we are most likely to have trouble. The case which Dr. McGuire has cited, where we had needle-like points which actually perforated the membranes, is a case in point. It seems to me that in a case of simple depressed fracture without any symptoms, unless the character of the injury is such as to lead us to believe that the internal table is driven sharply in, we can afford to wait.

The same may be said in regard to the fourth statement. There is some doubt as to the brain symptoms referred to.

In regard to intracranial hemorrhage, he says, "Trephine for the removal of clot and arrest of bleeding, when the probable seat of

hemorrhage is ascertainable and the clot is believed to be a localized one." If we believe that the hemorrhage was going on, and it was evident that the patient was going to die from the compression, it would be advisable to trephine; but if the symptoms were not urgent, it would not be wise to trephine, because the blood would certainly be absorbed if you give it time, and the only damage is temporary compression. If the hemorrhage is in the arachnoid space and produces compression, it is apt to be wide-spread. If it is within the dura mater it spreads over a large portion of the brain. If we could differentiate between hemorrhage, between the dura mater and the bone and that within the arachnoid, and we were confident that it was in the arachnoid, there would not be much prospect of removing it by operation.

I agree with Dr. Nancrede in regard to exploratory trephining. This gives a very wide margin, and I doubt if the author would be willing to permit the same liberty of action to others in his own case.

Dr. Charles T. Parkes, of Chicago, Illinois.

I am glad of the opportunity of speaking and expressing my pleasure in hearing this paper. The first proposition is one that I cannot agree with so far as to admit that the evidences of compression are due entirely to inflammation. I believe that in many cases of fracture the symptoms of compression are dependent entirely upon pressure from the displaced bone. These symptoms are often relieved immediately, as soon as this cause of irritation has been removed.

It is difficult to say in all cases upon what these symptoms of compression are dependent.

Dr. S. W. Gross, of Philadelphia, Pennsylvania.

Before we proceed any further we should like Dr. Roberts to explain what he means by "brain symptoms." Does he mean symptoms from compression of the brain or from inflammatory action?

Dr. Roberts. I can hardly explain the expression any differently from what I have done in the paper. Perhaps I have not made myself as clear as I thought I had. I say, "The complexus of symptoms, called compression of the brain, is due not so much to displacing pressure exerted on the brain substance as it is to some form or

degree of intracranial inflammation." I do not deny the fact which has been stated, that certain symptoms such as coma and paralysis may exist; they do exist. We call them symptoms of compression of the brain, but I mean to say that such group of symptoms, generally attributed to compression of the brain, are not really due to pressure caused by depression of the bone, are not due to the pressure of blood poured out beneath the bone. The investigations of Duret have shown that to have marked symptoms from pressure upon the brain you must have a considerable portion of the brain involved; and I believe, therefore, that these more or less limited depressions do not cause symptoms by pressure. If symptoms are present they are in most cases due, as Dr. Parkes has just said, to irritation, which, in my opinion, is the first stage of inflammation. Dr. Parkes has used the term irritation. When we say "compression," we, as a rule, should, in my opinion, say "irritation" or "inflammation." It has been used in the same way by Dr. McGuire.

Dr. Gross. We want an exact definition of "brain symptoms." Are the symptoms those which come on immediately after the injury, or after an interval of several days or weeks? We want to know what the symptoms are. We have not had the definition yet. Do you simply mean so-called symptoms of compression of the brain?

Dr. Roberts. I mean by brain symptoms those which are generally found after brain lesions and due to the lesion. I do not speak now, however, of symptoms which come from general lesions, and from lesions at the base. When I speak of fracture of the skull, with symptoms called brain symptoms, I refer to paralysis, coma, etc., of which Dr. Gunn has spoken as being caused by a local injury. My idea of brain symptoms in a depressed fracture are symptoms such as hemiplegia, coma, and stertorous breathing, which are usually attributed to depression of bone or to a clot upon the cerebral cortex. I refer to immediate symptoms; that is, symptoms occurring directly after the injury, and not those occurring several days or weeks after the injury, which all of us would attribute to inflammatory action. First, we have the shock of injury; then, the symptoms which are called immediate symptoms. It is these symptoms to which I refer.

Dr. Parkes. I cannot agree with the second proposition. I do not think that we have any right to give the patient even the

slightest increase of risk if the conditions are not such as to positively indicate the operation.

"The removal of portions of the cranium with the trephine or other cutting instrument, if properly done, is attended with little more risk to life than amputation of a finger through the metacarpal bone." I do not think that the two can be compared. They concern different parts of the body. As Dr. Nancrede has said, these fractures concern a part of the body that cannot be approached without danger at all times.

We all agree with the fourth proposition, but I dissent from the fifth, which is, that "perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons."

"Drainage is more essential in wounds of the brain than in wounds of other structures." It is not more essential, but it is more difficult to carry out. This was illustrated in a case which I may relate, and which shows the amount of injury the brain may receive without presenting many positive symptoms, and which presented a rare condition, that is, the occurrence of a flow of cerebro-spinal fluid from a fracture at the vault of the skull. I was unable to find a similar condition until I saw the last issue of Guy's Hospital Reports. In this case there were no symptoms of paralysis. There were fractures in two or three places, linear and depressed. There was a depressed fracture in the upper portion of the frontal bone, which was trephined and a large portion of bone removed. The membranes were apparently not injured. A drainage-tube was introduced and passed downward to the bottom of the cavity. There was no paralysis and no loss of sensation, but there was slight hebetude of intellect for a few days. On the fourth day after the accident, a large quantity of cerebro-spinal fluid flowed from the drainage-tube. This was the first case that I knew of in which such a flow occurred from the vault without apparent injury at the base. It was a mystery to me how it came. I do not see how irritation and laceration of the membranes should open the subarachnoid space so that there should be such a free flow of fluid. The fluid was noticeable on the pillows and bed. I could only explain it by the supposition that the injury had lacerated the brain into the ventricles. I see in a recent article in Guy's Hospital Reports, that Mr. Lucas reports three cases in which this accident was

proven to have occurred by the post-mortem. In these cases a fracture of the vault was accompanied by this flow of cerebro-spinal fluid. Here there was a good deal of laceration, yet there were no severe manifestations indicating the character of the injury.

In reference to injuries of the vessels, Dr. Roberts has referred to my case in which a piece of the superior longitudinal sinus as large as a coffee bean was scooped out. For a time I was at a loss how to manage it. Pressure controlled bleeding, but this could not be conveniently kept up. The idea then came into my mind to apply the same principles as in the treatment of wounds of other soft parts. I drew the edges of the opening together with three catgut sutures; the hemorrhage ceased and the patient got well.

In regard to the localization of the region of sensation, I have one case in illustration. A man was struck on the upper and posterior portion of the parietal bone with the muzzle of a pistol, producing a compound fracture perforating in character. I made an opening large enough to admit the finger. Both before and during the operation there was a large loss of brain-substance. My finger, when introduced, touched the tentorium cerebellum. I found there a foreign body which proved to be the sight of the pistol. Here there was a loss of the greater proportion of the area described by Dr. Roberts as the seat of sensation of the extremities, and yet the man showed no signs of loss of sensation except at the seat of injury. A large drainage-tube was introduced and the man was apparently recovering. At the end of two weeks, he was left in the hands of his medical attendant. Two weeks later, the patient had a chill, high temperature and trouble with one limb. I found that the drainage-tube had been removed. The wound was opened and a quantity of broken-down brain-matter and pus came out. The tube was re-introduced and the man recovered.

Dr. J. Collins Warren, of Boston, Massachusetts.

There is one thing that we must take into account in dealing with a depressed fracture of the skull, and that is the difficulty of getting successful drainage, as has been mentioned. Suppose we remove a fragment of bone and expose the soft parts, we have then the intra-cranial pressure to contend against. The contents of the cranial cavity bulge into the wound and cork up the hole that we have made. Moreover, we do not usually find a large collection of pus.

We have the evidences of irritation and inflammation without the accumulation of these products. When this intracranial pressure takes place, we have the pressure of the brain upon the sharp edge of the bone, even if no spicula have been left. We have then the chance of considerable brain injury after trephining.

It seems to me that there is another difficulty which meets us in considering these cases, and that is the difficulty of diagnosis. As Dr. McGuire has said we have to treat each case by itself.

I might cite one or two cases in point. Some time ago I was called to see a child with a depressed fracture of the upper anterior portion of the cranial vault. At first the child had no brain symptoms, but there was some febrile disturbance. At the end of a week the child became completely deaf. I did not interfere. At the autopsy a slight amount of depression of the internal table was seen, and pus was found in the fourth ventricle where it would have been difficult to reach by any superficial operation. The aspirator might perhaps have been introduced.

Again, in a hospital case of head injury in which the patient was unconscious and delirious for four weeks before he died, no evidence of brain lesion could be found. The cause of the injury in this case was unknown.

We may have so-called brain symptoms where the lesion is external to the brain. I have seen two cases this winter where it seemed to me that periostitis, and perhaps cellulitis at the back part of the head, had caused marked brain symptoms. It is true that there might have been a certain amount of concussion in these cases. They were both cases where the occipital bone had been hit, and there was a slight scalp wound. The first case got well after exhibiting symptoms which suggested the possibility of fracture at the base of the brain. There was no external lesion except great tenderness at the back of the head. The second case I was called to see in consultation. Remembering the previous case I examined the back of the head in the neighborhood of the scalp wound. At first there were no marked symptoms, but brain symptoms afterwards developed, and a large quantity of pus was found in the neck. After this was evacuated there was great improvement in the temperature and in the character of the mental state.

It seems to me that we have to take into consideration, not only the possibility of injury at the immediate seat of the blow, but we

also have to take into consideration that subtle condition known as concussion of the brain, whatever that may be.

In closing my remarks I should like to say something in regard to a case which illustrated very beautifully a local lesion of the kind which Dr. Roberts has so clearly explained to us. It was a case of a blow upon the head which I found in my wards when I went on duty the first of March. It had been examined by Dr. Walton, an able neurologist, and the various symptoms had been mapped out by him. The patient began with paralysis of the inferior extremities. You will see by looking at the diagram that the centre for the legs is at the highest part of the brain. Then came slight paralysis of the arms, and then paralysis of the lower part of the face on the same side. These symptoms were explained by Dr. Walton as possibly due to contusion or extravasation at the point marked "R" on the diagram, and that by gravitation the clot had worked its way down by the fissure of Rolando, producing slight paralysis of the arm and slight facial paralysis from involvement of the facial nerve, which is given off quite superficially. What further strengthened this diagnosis was that improvement took place in that order. As gravitation from the upper part took place the paralysis of the leg disappeared and the arm improved. There is now slight paralysis of the muscles of the lower part of the face, with complete return of motion in the inferior extremity, and a partial return in the upper extremity.

Dr. L. McLane Tiffany, of Baltimore, Maryland.

I think that there is scarcely anything to be added to what has already been said. In regard to the conversion of a simple fracture into a compound one not being attended with increased risk, I know that it is the fashion to say so, but I doubt its correctness. The cutting of tendons is still done subcutaneously. A fracture anywhere in the body is never opened unnecessarily. Most of us prefer that wounds shall become subcutaneous as soon as possible.

As regards the second and third propositions, certain monkeys have been cited as having their heads opened without bad results, and in these cases there was no other injury. The operation is discussed as though there were no other lesions than that produced by the operator. There is in these cases associated injury of the brain and its membranes. As regards the third proposition, to compare

trephining of the skull for a lesion previously inflicted with amputation through the hand, I think is not possible.

In the majority of cranial fractures the inner table is more extensively shattered and splintered than the outer table. (?) That is true provided the injury comes from the outside, but if the bullet perforates the skull from within outwards, where it comes out will be the most fractured. If an individual discharges a pistol in his mouth or beneath the chin, and the bullet passes through the skull, the outer table will be more splintered than the inner.

"Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons." On the other page we have only two cases where trephining is not to be done; and it would appear that they are reserved for the diagnostic operation. The writer has left us in doubt where the exploratory operation is to be done.

I now come to the large trephine. It had a suspiciously new and unused look! The writer of the paper suggests that the trephine should be half a circle. Why not use the ordinary trephine and apply it twice? The multiplication of instruments is a mistake. If an instrument in use is sufficient, it would be better to employ it.'

There is an omission in the paper which should be corrected. There should an absolute, hard and fast line drawn between fractures in childhood with depression and fractures in adults. I have no doubt that every one within sound of my voice has again and again seen injuries in childhood for which they would have instituted operative measures in adults I have on three or four occasions seen markedly depressed fractures in childhood and have let them alone—of course I refer to simple fractures. The children have done perfectly well. Only three months ago, I saw a case in which there was a depressed fracture in the upper anterior parietal region, in which I could lay my finger in the groove. The depressed bone is gradually resuming its normal position.

There is still another distinction to be made which I think will not be appreciated by all present, and that is the difference between the negro and the white race. In the negro inflammatory symptoms are not so marked, and they do not run so acute a course as in the white race. Shock after an injury is infinitely less than it is in the white race. I have watched this in a general hospital, and I come from a region where the colored race are constantly under

treatment. There is no doubt about the different course which inflammation pursues in the two races.

There is another small point. Under the head of treatment, No. 9, we find "No evident depression, no brain symptoms." How is that diagnosis of fracture made? I think that it is impossible. Again, No. 5, "No evident depression, no brain symptoms." I think that is a physical impossibility to produce a comminuted fracture under such circumstances.

No. 2, "No evident depression with brain symptoms. Incise scalp and trephine." Where would the trephine be applied? The diagnosis I think could not be made. Suppose the injury is in a position where we know little about the centres. It might be a fracture by counter-stroke, or there might be extravasation of blood on the opposite side of the brain. An injury on one side may produce an injury of the same side or of the opposite side. I make these remarks not in a spirit of criticism, but I think these as well as other points should be considered in a paper so exhaustive.

Dr. Gross. In reference to the last point referred to by Dr. Tiffany, I know that there may be a fracture of the skull without evident depression and with brain symptoms. In the army it was common to have a linear fracture without depression with brain symptoms. Some years ago I wrote an article on this condition as found in army cases. There may be a linear fracture and the dura mater may be separated from the bone, and so many small vessels may be torn as to give rise to effusion of blood with subsequent symptoms of compression, which come on more slowly than after an injury complicated by laceration of the anterior branch of the middle meningeal artery.

I think that there is a consensus of opinion in regard to many of the sweeping views adopted by Dr. Roberts. I do not believe that any one regards a compound fracture as being so simple an affair as a closed fracture; I do not think for a moment that, even with the improved methods of treatment, a compound fracture is as innocuous as a closed fracture.

I do not believe that trephining is as safe as amputation of a finger. The tables of Dr. Amidon, published in the last number of the *Archives of Surgery*, will not bear a critical examination. He has a theory, and he has made the facts fit the theory.

To carry out the antiseptic method of treatment in wounds of the

skull is difficult on account of the mobility of the head and the tossing about of the patient.

Drainage is more important in injuries of the brain than in ordidinary wounds, simply for the reason that we want to get rid of pent-up fluids as soon as possible, because these interfere with the proper performance of the functions of the brain. It is exceedingly difficult to drain the brain. In the case described by Noyes some years ago in *The American Journal of the Medical Sciences*, an opening was made at the vault of the skull and a tube passed through, the patient recovering. If we simply pass a tube into the wound the drainage is not thorough. I think drainage should be carried out here as it is in wounds of other structures.

In regard to divisions, B, C, D, and E, all are agreed. In the removal of tumors more experience is required.

In the treatment of gunshot injuries of the skull I presume that the proper plan is to enlarge the opening and extract the fragments of bone, and then with the finger or a proper probe try to follow and remove the missile and any fragments of bone that may be there. If it is a homicide case, the attorney says that the patient dies because you did not make a thorough examination, and that had you made such examination and removed the foreign bodies, the patient would have survived. Again, if you are called to a case and do explore the wound, and the patient dies, the man's sentence is commuted because the surgeon killed the man. This happened in the recent trial of Brotheras in the State of Pennsylvania.

I wish that we could enunciate some line of treatment of gunshot wounds of the skull. We should not hesitate in such injuries of the brain to enlarge the opening and follow the track of the ball with the finger or a proper probe, because it is essential to remove foreign bodies from all tissues, and especially so from the brain. Although patients may recover where the offending substance has not been removed, yet they are apt to suffer subsequently from abscess, softening of the brain, or epileptic affections. I esteem it of great importance to carry out the same principles of treatment in gunshot injuries of the brain as apply to gunshot injuries of other tissues.

A most extraordinary case has been recorded in the *New York Medical Record* by Fleurer. A man was admitted into the hospital with a gunshot injury of the skull. He followed the track of the bullet, and found that it had impinged upon the opposite side of the skull. He trephined at this point, and found that after the ball had

struck it had dropped, and it was discovered half an inch below the trephine opening and removed. The man got well. This case illustrates the importance of carrying out the same principles of treatment in wounds of the brain that we adopt in wounds of other tissues or organs.

Dr. Edward M. Moore, of Rochester, New York.

I do not rise to discuss the paper directly, but rather to continue what Dr. Gross has said. It is cognate to Dr. Roberts's paper. This seems to me to be one of those things on which we should give a decided expression of opinion in regard to the treatment of gunshot injuries of the head. A man puts a pistol to his temporal region and fires a bullet into his brain, the question is, What is to be done? The satisfactory termination of the case which Dr. Gross relates is extremely gratifying, although I have not met with such favorable results. The case brought before the courts to which he alluded I had noticed.

If there is one thing which we have carried out in surgery with benefit, it is the successful drainage of wounds. I am quite sure that it is a great advance. The use of drainage-tubes in wounds of the brain is surely a modern idea, and the cases mentioned are very gratifying.

In those cases which I have seen lately, I have carried down a large probe along the track of the ball, and I have uniformly found that the bullet traversed the whole brain, going to the opposite side, usually with force enough to pass at a reflected angle a little away from that point. In one of the cases it was half an inch from that point. In my last case I carried the probe and trephined, and fortunately hit the exact point where the injury had been produced. I carried in a tube, but did not carry it through. I desisted. I was wrong; I should have carried it through. It seems to me that this is the plan to be pursued.

The successful case reported is certainly extraordinary. We all know of the Vermont man who had a bar of iron driven through his head. That man's recovery could only be explained on the ground of the perfect and free drainage which took place. In a case of injury of the skull over the longitudinal sinus in a child, I cut down and applied the trephine. The child recovered without a bad symptom.

We have these extraordinary cases, and this leads me to make

another remark, and that is in reference to the unreliability of statistics in regard to trephining. The cases are so unlike, that statistics have not the value that they have in other cases.

Dr. David Prince, of Jacksonville, Illinois.

I have not much to say, but I am in general accord with the views expressed in the paper. From being one of the most dangerous operations, trephining has, with antiseptic methods and management, become one of the safest operations. I am old enough to have seen the difference in the behavior of wounds of the cranium, whether accidentally or purposely produced, under the old management and under the new. Since the employment of methods to prevent putrefactive action, I have seen no cases of hernia cerebri or similar conditions.

The comparison was made in the paper between the operation of trephining and amputation of the finger, and one considered as safe as the other. This is probably an exaggeration growing out of enthusiasm, but I think it may safely be said that it is as easy to prevent putrefactive changes in trephining wounds as it is in amputation of the finger by those antiseptic precautions now so familiar. But if through neglect or through a depraved condition of the system of the patient, such changes do occur in the finger, they do not necessarily endanger life. If these changes occur in a trephining wound loss of life is almost certain. That is the difference.

I may illustrate this by a case recently under observation. A man was injured by being struck on the forehead with a brick thrown by his angered son. This produced a compound comminuted fracture. Trephining was of course the proper treatment. The operation was performed several hours after the injury, and the attempt was made to secure carbolic acid wet dressings. The man was wilful and unreasonable, and fought against treatment, and being in a country house treatment could not be enforced. The consequence was that septic changes occurred, and the man died from septic meningitis. If this had been an amputation of a finger this accident might have occurred and, although unfortunate, the man might have recovered.

These processes enable us to resort to trephining as an exploratory operation, as has been so well exemplified by the paper, without the fear of doing harm, which under the old management would necessarily have been imminent.

The conditions of treatment are precisely the same as in injuries of other parts. If we carry the patient through shock and hemorrhage, and secure those antiseptic arrangements not necessary to detail, there is one condition which it is necessary to maintain, and which is not only not mentioned in the paper but in regard to which the paper is misleading in its comparison of trephining to amputation of a finger, and in regard to the ability to go home. That condition is rest. The importance of rest should be emphasized rather than belittled. Rest to the utmost extent should I think be enjoined.

Dr. Prewitt in his remarks made a comparison between injuries caused by the process of trephining in closed fracture of the cranium and opening of the leg in compound fracture of that member. I think that the cases are precisely parallel with this difference, that, in the head, the circulation is more active, and therefore that the process of repair will go on more rapidly than in the leg. In the leg we have no occasion to convert a simple fracture into a compound one, except it be in some rare cases where it is necessary to secure the reduction of misplaced fragments.

In regard to the use of the trephine in the treatment of epilepsy I would call the attention of the Fellows to one case in which, under the old methods, the brain escaped any condition of inflammation following an operation in which a three-quarter inch trephine was used and exostoses removed. This was in 1860. The patient recovered without any inflammatory symptoms whatever. Now, as I recall the case, there were adhesions of the dura mater around the circumference of the trephine hole, so that the way was shut to the encroachment of septic agencies. If the operation had been performed in a case where there were only the ordinary attachments the result might have been different. By the use of carbolic acid and other antiseptics we can guard as well against septic agencies as if there were adhesions, as in the case referred to.

I operated once for insanity following an injury with negative results. The advance of knowledge upon this subject, as indicated by the paper and the discussion of it, leads us to felicitate ourselves and mankind upon the probability of saving many lives, which under the old management would have been lost.

Dr. J. H. Brinton, of Philadelphia, Pennsylvania.

Before the discussion on this subject is closed I desire to say a

few words, and afterwards to ask a question. First, in reference to the leading proposition in the synopsis of Dr. Roberts's paper. "The complexus of symptoms called compression of the brain is not due so much to displacing pressure exerted on the brain substance as it is to some form or degree of intracranial inflammation." Is this statement altogether true? Is it not calculated to carry with it some misunderstanding? Clinically speaking, when called to a case of this kind, do we not first of all examine as to the existence of depressed bone, producing as it so often does symptoms which we desire to relieve by its elevation? It seems to me that in the great majority of such cases the displaced fragments first attract our attention, and it is to them, and their rectification, that we direct our immediate surgical treatment.

I do not wish to take up the time of the Association with the report of cases, but I would like to refer to one which I saw some years ago. The patient was a boy seventeen or eighteen years of age, who received a remarkable fracture extending from one external angular process of the frontal bone to the same point on the opposite side. One edge of the fractured arch closely overlapped the other. I saw the lad a short time after the accident, he was then in a state of absolute coma, and I at once took the proper measures for elevating the bone. I sawed off a long narrow transverse edge of bone with a Hey's saw, and then, after a slight attempt at elevation, the bone sprang into place with an audible snap. Almost instantly the patient's senses returned, and he emerged from his condition of coma. Here was undoubtedly a case of compression of the brain due to the pressure of bone, with very little extravasation of blood. It was a case in point, and in view of such cases I do not think that we are justified in rejecting the idea of pressure as a factor in producing the complexus of symptoms called compression.

I do not allude to many of the other points which have been discussed. I believe in the early use of the trephine, but cannot look upon trephining as a trivial operation. It is a serious procedure, and I do not see how we can regard it in any other light, if we consider the consequences which must ensue from the admission of air into the intracranial cavity, and if we reflect upon the cerebral contusion and laceration which so often accompany injuries of the head. It is only necessary to recall our observations in the post-mortem room, or to glance at the exquisite plates of Mr. Jonathan

Hutchinson, to understand how, as a result of trephining, the air and all it carries with it may penetrate into the interior of the brain.

Remarks have been made in regard to the lateral ligature of the sinuses of the brain. In the case of a child kicked by a horse a fracture was produced over the lateral sinus. Here I had no difficulty in ligaturing the wall of the sinus laterally by raising it with an artery forceps. The difficulty disappears when a sufficient amount of the bone is removed.

The question I would ask is concerning the treatment of fractures of the skull. What have we done yesterday and to-day? Have we made any advance in suggestions for the treatment of these injuries so elaborately classified in the paper under discussion. It would appear that we are all agreed as to cases of compound fracture of the skull with depression, with or without symptoms. Here there is no difficulty in the treatment—trephining and elevation. The only case where there is any doubt is in that of simple depressed fracture, with few or no symptoms. Under these circumstances we have to decide what to do. Without operative procedures, such a patient may recover entirely, or he may recover for the time being only, and traumatic encephalitis may ensue; or at a subsequent period, more or less remote, he may suffer from all the horrors of deferred irritation of the brain or from epilepsy. What is the treatment? I believe that this can be briefly summed up, and indeed it has already been in a few lines.

Let me quote the teachings of the founder of our Association, who, in his System of Surgery, in speaking of the treatment of simple fracture with depression of bone, says: "That where the fracture is of small extent, free from comminution and without much depression, it had best be let alone;" but he adds, that "Where the bone is forced down considerably so as to impinge very decidedly upon the brain, or if it be comminuted or jagged at the edges the sooner it is raised or removed the better; since, if it be allowed to remain, it cannot fail to become a source of trouble, either by exciting inflammation, or by causing serious secondary effects. I am fully, indeed painfully, sensible of the responsibility which I incur in giving this advice; but I feel satisfied after mature consideration, aided by the light of experience, that it is the best, if not the only proper course to be pursued under the circumstances. A man laboring under such an affection is never free from danger; he may get well, or be well to all appearances, and yet be only

partially cured, subject at any moment to have his mind and life imperilled by the broken bone. It is like the sword of the tyrant suspended over the head of his subject."

Here we have the whole subject in a nutshell, and here is the true plan of treatment to be followed in these cases. Such words of wisdom and experience seem to me to be all-comprehensive; to them, little can be added, and from them little can profitably be taken away.

Dr. Theodore McGraw, of Detroit, Michigan.

I wish to take exception to a statement made yesterday, that compression in itself was not a very bad thing. The opinion was advanced, as I understood it, that the brain would accommodate itself to the compression. That is undoubtedly true in childhood, but I do not think that it is true in adults. It is not often that we get the chance of seeing a case of uncomplicated compression after a considerable lapse of time. I had one such case which I trephined. It was that of a negro twenty-four years of age, a painter by occupation. He fell from a scaffold and received a very curious circular depressed fracture, just as if it had been pressed in. It was perfectly smooth. At first he was stunned, but afterwards recovered, and attended to his work, and apparently suffered from no symptoms of cerebral irritation. Later he began to be dizzy, and this increased to such an extent that he was liable to fall, and was compelled to give up work. He then came to the Harper Hospital in Detroit to be operated on. I trephined over the middle of the cup-shaped depression. As I lifted the button of bone, the dura mater bulged into the wound. The portion of bone removed was smooth. The dura mater presented no evidences of irritation. From the day of operation the man had no more giddiness. This was a case of uncomplicated compression. There was no irritation, except that produced by the compression. In this case the brain did not get accustomed to the compression. The operation was done a year after the accident. The last I heard of the man he was painting on scaffolds four and five stories high.

Another point. Dr. Nancrede said that after irritation of the brain had begun, the operation was useless, except to take away irritating spicula of bone.

Dr. Nancrede. I said that it could only remove the secondary cause. It could not remove the inflammation already set up.

Dr. McGRAW. Suppose you have a felon on the finger. There is irritation. You lance it and remove the tension. When you trephine the skull for inflammation of the brain you remove the spicula of bone, and you remove the tension, and this relieves the inflammation.

Dr. P. S CONNER, of Cincinnati, Ohio.

One or two points brought out yesterday may well be considered. We are in accord that in a large proportion of skull injuries, removal of the broken pieces of bone, especially if there is an open wound, is the proper practice. If, in a case of depressed fracture without open wound, we take away the depressed bone, we give the patient a chance of avoiding the conditions which may follow. I do not believe that the operation of trephining is a trivial one. If we know anything, we know that it is attended with a considerable mortality. The cases which give us the nearest approach to the true mortality of the operation are those in which it is done for epilepsy. There the mortality is about ten per cent.

We know that the worst consequences follow fracture which injures the external table but little, but which produces great damage of the internal plate. In such a case the chances for perfect recovery are better after removal of the damaged bone. It hardly seems to me that the addition of a wound of the soft parts adds materially to the gravity. While this is a matter of some moment, it is of but little if we can by it prevent the after consequences of such injuries. There is nothing more fatal than a meningitis developed by a sharp point of bone. Almost every case, if not every case, so affected dies.

It is not the part of wisdom to leave the patient exposed to such a risk.

As said by Dr. Tiffany, the conditions are different in children from what they are in adults. We can temporize in the case of children, while we cannot in the case of adults.

I was surprised at the expressions of confidence in regard to the operative treatment of gunshot wounds. I have seen many cases of this injury. It is not a trifling matter, at least for an inexperienced operator, to be forcing probes in different directions through the brain. If we are to believe some writers, it is not the presence of the ball that does the damage; this adds little or nothing to the gravity of the case, nor do the sharp pieces of bone do any damage.

It is only the entrance of germs that cause the trouble. I do not think that it is the part of wisdom to be trying to follow up the track of the ball, to scoop out the damaged brain-substance, and attempting to put drainage-tubes through. If a track has been made through which a drainage-tube can be passed well and good.

I was much interested in the case described by Dr. Brinton in which there was compression from depression. As a rule a very large amount of the vault of the cranium must be depressed in order to produce compression. It has been estimated that the depression of one-twelfth to one-eighth of the whole surface is required. We occasionally see, as in Dr. McGraw's case, a very limited depression followed by compression. In most of these cases a careful examination will show other conditions, such as blood-clot. Under the age of eighteen or twenty there may be a considerable amount of depression without any injurious consequences whatever. I do not believe that it is either safe or wise to declare that trephining should be done in all these cases of head injury. It is not wise to regard it as a trivial operation. There is a certain amount of damage done to the bone, and if inflammation of the diploic veins is produced, there may be serious consequences.

There are a good many points that are still unsettled, and while I believe that in open wounds attended with comminution and depression, the operation should be done at once, I also believe that in the majority of simple wounds, with a moderate degree of depression with no symptoms, we should simply wait and watch.

Dr. N. P. Dandridge, of Cincinnati, Ohio.

I wish to report a single case bearing on this point. It is that of a German injured during the riots. He was struck on the right side of the head with a glancing ball, which made a scalp wound four inches in extent in the median line. He was carried to the hospital in an unconscious condition, and was afterwards taken to his home. I saw the case six days later. The parietal bone was bared, and there was a linear fracture without any depression. The symptoms at this time were paralysis of the muscles of the forearm of the opposite side, and frequently recurring spasms of the left side of the face. These occurred at very short intervals without unconsciousness. The man had not fully recovered his intellectual powers, although he could answer questions. When left to himself he had slight muttering delirium. At this time he could not recollect what

had happened between the time of the accident and three days later. We had here a case of compound fracture over the motor area, followed by paralysis of the muscles of the opposite side of the forearm. These are the symptoms we should have expected from our knowledge of localization.

I saw the man at this late period, and as the symptoms were not increasing, and there had been, indeed, a slight amelioration, it was decided not to attempt any interference, but I confess that I felt some uncertainty as to this being the proper course. In two or three days the spasms ceased and consciousness was restored, and the man made an uninterrupted recovery. The paralysis disappeared in three or four weeks, leaving behind a slight numbness of the little finger. This still exists after the lapse of a year. This case is of importance as bearing upon certain points in the discussion. We had a compound fracture followed by paralysis which corresponded exactly with the seat of injury, and yet these symptoms all disappeared without any interference. If I had seen the case shortly after the accident I should have been disposed to trephine.

In regard to brain symptoms as connected with compression, I do not think that sufficient weight has been placed upon the different degrees of symptoms which we are to expect from the different location of the injuries. If we wait for symptoms in fracture of the frontal bone, we shall have to wait until a fatal degree of inflammation is developed. Where the injury is over the motor areas we may expect symptoms of paralysis to result. It seems to me that in all these cases after restoration to consciousness has occurred from the acute effect of the injury, that the question of brain symptoms is to be decided largely by the position of the injury.

Dr. B. A. Watson, of Jersey City, N. J.

I have no desire to discuss this paper, but there is one question which has been raised by Dr. McGraw, and that is the accommodation of the brain to compression. I have a case in point. A police officer, three years ago, received a fracture of the skull at the anterior inferior angle of the right parietal bone. He was brought home in an unconscious condition, and I saw him about twelve hours after the injury. He was still unconscious, and could not be roused. I was able to distinctly locate the fracture, although it was simple. I decided to leave it alone. The man suffered nearly three weeks with acute delirium, but gradually the delirium dis-

appeared. There were other symptoms in addition to the delirium, showing that the brain was seriously involved. He lost memory, and was unable to talk coherently for three weeks, but notwithstanding this he recovered. I saw him a year and a half after the injury, and there was still marked depression. This was so marked that there could be no mistake about it. This man has recovered, and can perform his duties in a satisfactory manner. It is true that alcoholic beverages act on him more readily than formerly. This man was forty years of age. While in young children the brain accommodates itself readily, still even in adults it will accommodate itself to considerable pressure.

The PRESIDENT. I have but few remarks to offer on this subject. It has always been one of the vexed questions in surgery, and is still far from being settled. The paper of Dr. Roberts is a most excellent one, as is evinced by the lengthy and spirited discussion it has elicited.

There are two classes of cranial injuries, which are separate and distinct the one from the other. In the one the effects of the injury are not limited to the cranial walls, but extend deeply into the brain substance, and even in some cases to the base of the skull, as when the injury is inflicted by falls from great heights, or when heavy substances fall on the cranium from heights. Under these circumstances the operation of trephining could not possibly remove the effects of the injury inflicted, and would, therefore, be useless if not injurious.

In the other the injuries are local, being confined to the bone itself, or extending but slightly beyond. In these cases most frequently the bone is broken and pressed down upon the membranes and the brain. Oftentimes sharp spicula are driven upon or even into the meninges and brain. It is in such cases that the trephine is demanded, and it is only in such that great benefit is likely to follow its use. The sole object of the trephine in fractures of the skull is to remove bone, that by its changed position is pressing upon or driven into the contained organs.

For the relief of the symptoms of compression of the brain caused by the depression of fractured bones the trephine is certainly not indicated. The brain is capable of accommodating itself to any amount of fixed pressure, especially if the depression is broad and smooth, and the symptoms will soon disappear. Besides, there are

no symptoms that distinguish with certainty compression from concussion of the brain. In both cases the symptoms arise, doubtless, from disturbance of the vascular or nervous circulation, or from some molecular change in the brain-tissue. In many cases it is impossible to tell if they are due to the one or the other; nor does it matter much, for in either case if there is no serious contusion or laceration of the brain the symptoms will soon pass away without operative interference.

When the symptoms of compression are due to extravasation of blood the pressure is prolonged, is increasing constantly, and may produce death by the pressure exerted upon the encephalic mass. In such cases, if the location of the clot can be determined either from the character or position of the fracture or from symptoms arising from its interference with special parts of the brain, the trephine is indicated. It is employed, however, as a *dernier ressort*, for in a large number of cases the condition cannot be remedied.

When compression is due to the formation of pus within the cranium the same rules should be followed that guide us in the evacuation of pus in other parts of the body. The trephine should be used for its escape as the lancet is used in the soft parts.

The principal and only use of the trephine, in my opinion, is to remove points of irritation produced by fragments and spicula of bone, which press on the surface of or perforate the membranes, and act as constant sources of irritation to the brain.

The first symptoms that follow an injury of the head are usually those of compression or concussion of the brain. These symptoms generally pass off in a short time, but are oftentimes followed by those of irritation of the brain or its meninges, characterized by the peculiar position of the patient, who is seen lying on his side with his body curled up and limbs flexed, like a rabbit struck on the head (as you have often seen in your hunting days), in a semi-conscious state. The arm on the opposite side is often in a condition of tonic contraction, sometimes tremulous, or even affected with spasms, the muscles of face often twitch, the pupils contract, and the eyes are peculiarly sensitive to light, etc. etc.

When these or other symptoms indicating irritation of the brain are present, I think that it is the imperative duty of the surgeon to operate at the earliest possible moment to prevent that which cannot be cured if the pathological changes progress to a greater extent. Trephining is only useful for preventing that which, when it occurs,

cannot be cured. I think that the trephine should be limited to this, and this alone. It can remove points of irritation, and that often stops the progress of inflammation. If inflammation has set in, the trephine cannot, in my opinion, do any good, except in so far as it removes the cause of inflammation. The removal comes when it is probably too late. The large majority of patients will die under these circumstances. The trephine is to be used as a preventive, and for that purpose alone. That is my idea in regard to the trephine.

There are other points to which I shall call attention. In regard to the conversion of a closed fracture into a compound one by incision. This is a question which I think can be easily settled. None of the gentlemen seem to have taken into consideration the difference between the bones of the skull and long bones. They have compared fractures of the skull to fractures of long bones, and have asked the question: What surgeon would open a simple fracture of an extremity? None of us would like to do it. In the cranium the bones are freely nourished, with very little cellular structure—with no medullary cavity. There are no loose cellular tissues around it. These bones are not at all similar to the long bones.

It has been my practice, wherever there was any doubt in regard to the nature of the injury, to make a free incision to the bone. I have done this in a number of instances, and have never had cause to regret it. It has never been followed by bad consequences.

In reference to the danger of trephining the skull. That depends on circumstances. In the operation of trephining, *per se*, there is no danger. If the trephine is used carefully, the simple cutting out of a portion of bone and the removal or elevation of broken fragments cannot be dangerous. The danger is not in the trephine. The injury to the skull is frequently associated with injury to the brain or membranes, and under these circumstances the operation may be followed by death. It seems to me that the careful removal of a portion of the skull cannot involve any great danger. I do not consider it as devoid of danger as cutting off a finger; I regard it, however, as a very safe operation.

As regards the fourth proposition, all are agreed.

In regard to the fifth, I do not know that we are. Perforation of the cranium as an exploratory measure should be done guardedly, and not unless symptoms point to irritation of the brain which can be located. It sometimes happens that while the outer wall of the

skull has not been broken, the inner table is fractured and a number of spicula driven into the brain. If symptoms indicating a high degree of irritation follow a blow on the skull without external fracture, the probabilities are, in nine cases out of ten, that the internal table is fractured, and it is then the duty of the surgeon to make an exploratory operation to discover whether or not the internal plate has been broken. The danger will be less than to leave the fragments of bone pressing upon the brain. Under no other circumstances would I resort to exploratory trephining in fractures of the skull.

Drainage is essential.

In regard to hemorrhage, especially from the sinuses. Several Fellows have spoken of laterally ligaturing or sewing up the sinuses. On one occasion I was so unfortunate as to thrust the point of the trephine into the longitudinal sinus; I put my finger on the opening until I applied a piece of lint imbued with an anti-inflammatory fluid. This simple pressure arrested the hemorrhage without trouble. On another occasion the sinus was torn by a fragment of bone, but slight pressure controlled the bleeding. I do not think that it is necessary to resort to ligature of the sinus, because the bleeding can be so readily checked by pressure.

Dr. ROBERTS. There are a good many points which have been brought up, to which, on account of want of time, I shall not attempt to reply. I have been gratified by the discussion, because it has served to convince me of the truth of the propositions as stated. I worded these propositions in such a way as to show that I did not mean to say that in every case such a thing is so, but that in the majority of cases it is so. I say "the complexus of symptoms is due not *so much* to displacing pressure," that "the conversion of a closed fracture into a compound one is attended with *very little* increased risk to life," showing that I admit that there is some increased risk, and so on.

The views which I have brought forward to-day will be more firmly believed in four or five years hence than at the present time. Dr. Gunn's opinions were opposed three years ago, but are not so strongly opposed to-day. I shall not be opposed five years from this time as vigorously as I am to-day.

A difference must, in my opinion, be made between the words compression or squeezing and pressure. Where there is depressed bone there is pressure, but there is not compression or squeezing of

the brain. We may speak of the brain and spinal cord as a mass of jelly in a bowl, and a connecting tube running downwards from it. If a depression of a limited portion of the skull is made, as in an ordinary fracture, the brain cannot be compressed or squeezed. It will be pressed upon, but the fluids are merely displaced, and run into the spinal canal, so that the amount of pressure is not materially increased. You may squeeze the brain by putting it in a vise, or between the wall of a house and a wagon, or by vast crushing fractures of the skull, but compression or squeezing of the brain is, in ordinary cases of fracture, impossible. I wish especially to make the distinction between compression or squeezing and mere pressure. The brain is too soft to be squeezed by such depressions as ordinarily occur.

It has been stated that we sometimes have such symptoms as hemiplegia, coma, stertorous breathing, and so on, coming on immediately after injury, and I have been asked whether these are not evidences of compression. In most of these cases, the symptoms are due to shock, to laceration, to contusion, or, possibly, to that obscure condition called concussion, which, by the way, I do not believe in as a functional condition. It should be translated minute and multiple laceration. The nerves of the dura mater are involved in all these cases. The branches of the trigeminal may have something to do with the development of the symptoms. Such is the causation of some of the immediate symptoms. Inflammation probably causes the other. It has been said that inflammation cannot develop so rapidly as to account for these symptoms. If you put your finger into my eye and make pressure, you do not compress the eyeball, but in half a minute there will be evidences of inflammation. So in the brain, inflammation may develop rapidly. The immediate appearance of symptoms does not mean that the brain is squeezed. If the brain were hard enough to be squeezed, we should seldom have subcranial hemorrhage, for it takes very little pressure to check bleeding; but as the soft brain is easily displaced room for hemorrhage is afforded and bleeding between bone and dura occurs.

Dr. Prewitt has referred to the escape of blood between the membranes, and he has said that compression results, and that in a short time the blood will be absorbed and cure effected without operation. I am inclined to doubt this reasoning. The blood would produce some pressure, and if cure took place it would be by absorption of the blood, but would probably not be a complete cure.

Pachymeningitis and other conditions may follow the non-absorption of such blood-clots.

Dr. Tiffany has said that it was better to have a closed wound than to have an open wound. As a rule, closed wounds are better than open wounds, but open wounds are better than closed wounds which have inherent dangers connected with them which cannot be recognized without opening them.

It has been said that we cannot compare the operation of trephining with amputation through a metacarpal bone—that such a comparison is impossible. It is unwise for any one to say that a thing cannot be done. The comparison has been made. I have taken the statistics of Ashhurst, which have been collected from many sources. I took first the statistics of amputation in front of the metacarpal bone, and then the statistics of amputation through the metacarpal *bones*, which is a more serious operation. I then took the average of these as giving about the correct mortality for amputation through a single metacarpal bone. This gives four or five per cent. In the statistics of Dr. Amidon, the cases of operation on the skull are open to inspection. Dr. Gross has objected to them, and I admit that Dr. Amidon is not a surgeon, and may have given the statistics a more favorable interpretation than a surgeon would be apt to do. I have taken these statistics, and put down the mortality of trephining, uncomplicated with other injury, as about three per cent. Now, in order to neutralize any possible error of Dr. Amidon, let us double this, and say the mortality is six per cent. Comparing this with four or five per cent., the mortality for operations through a metacarpal bone, I find that my original proposition still holds good. It reads, "The removal of portions of the cranium by the trephine or other cutting instrument is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone." It holds good, you will see, even if Amidon is wrong in half his cases.

It has been asserted that the question of which table is the most splintered, depends upon the direction in which the ball goes. If the ball comes out of the vault of the skull there will be more splintering of the outer table. This point is discussed in the paper, but was omitted for want of time.

In regard to exploratory trephining in obscure cases. I was asked if I would be willing to have my head trephined in an obscure injury. I unhesitatingly say yes. In such a case I should want my

skull trephined at once, and not be allowed to die from spicules of bone pressing into the brain. Every conscientious surgeon should put himself into the place of the patient, and it is a frequent statement with me, when speaking to patients concerning operations of various kinds, "If I were in your place, I would have such and such an operation done." I should prefer the operation of trephining to obscurity in the diagnosis in my own case; hence I should recommend it to others.

There seems to be no disagreement in reference to drainage, except as to the method to be employed.

Dr Gross has referred to Fluhrer's case; and has said that it was reported in the *New York Record*. The reference should be to the *New York Medical Journal*. It is referred to in my paper. The case was reported some three weeks ago. I think that we owe to Fluhrer more than we do to any one who has published anything within the past few weeks, because the case was treated in a scientific manner throughout. The man was placed in such a position that the orifice of the wound should be vertical. A probe with a large ball on the end was then taken and inserted into the wound, and allowed to follow the track by gravity. It was not pushed into the brain at all. It was found that the probe went to the posterior part of the skull. The trephine was applied over this spot. There he found signs of extravasation, but the bullet had been reflected. The head was put in such a position as to make the second opening vertical, and the probe again introduced and found to go in a certain direction, and then I think he trephined a second time. This is a reasonable way of tracing the course of a bullet. He then extracted the bullet, and instituted drainage. He did this in rather an ingenious way. First, a thread was passed through, then something a little larger. Drainage was continued for a long time, and finally the patient was cured. The case should be read in full by every Fellow of the Association.

Bullets may become encysted, and who knows but that they may work downward from gravity and rest upon the important structures at the base of the brain, and produce death secondarily because the surgeon lacked sufficient force to apply to the brain those surgical principles which he applies to other portions of the body.

In regard to cerebral location a case is reported in which the posterior part of the occipital lobe was interfered with without the presence of sensory phenomena. The difficulty was that the injury

was behind what I described as the sensory region. This is in the parietal, not in the occipital region.

In all cases where you desire to operate after a diagnosis by cerebral localization, you must eliminate all deep-seated lesions and all local injuries as referred to by Dr. Nancrede. The symptoms due to dura mater lesions must also be considered.

I do not advocate the use of half a trephine, because a big trephine is too big, as seems to be inferred by one speaker. I advocate the trephine cutting on half its edge, so that when you have got through the skull in one place it may be used, thus lessening the risk of wounding the membranes. In ordinary trephining most of the instruments sold are too large. Where you only want to elevate, you want a small trephine, but when you are searching for lesions located by cerebral symptomatology you need a large trephine.

As has been said, there is a good deal of difference in children and in negroes. Not long ago, I saw in consultation, a negro who had a fracture in the occipital region. There was a depression; but, I said to myself, "Here is a negro with an injury of the thickest portion of the skull, and he belongs to a race that has thicker skulls than most people; hence it is probable that the depression is limited to the outer table, and does not involve the inner table." I advised against operation, and the man recovered.

In regard to children, I might report a case of a child kicked by a horse. There was a soft place in the head indicating the position of a simple comminuted fracture. There was not much depression. Pressure upon the fracture caused a flow of blood and air from the ear. Some years ago that would have been a case in which I probably would not have operated, for here was apparently a fracture at the base of the brain involving the petrous bone communicating with the ear. It did not seem that the air came from a fracture involving the mastoid process; and besides in a child the cells in this bone are not developed. Here, owing to my opinions, I made an incision over the depressed portion, which was as large as a silver dollar, and removed some pieces of bone, and the child promptly recovered. He would almost certainly have died if let alone; and such would have been the treatment I think by many practitioners.

I do not say that trephining should be done in all skull fractures. I stated that there were one or two conditions in which trephining

should never be done. Then, there is a series of cases in which trephining should possibly be done; then, a series in which it probably should be done, and then there is a series in which trephining should always be performed. I agree perfectly with Dr. McGuire, that it would *not* be wise to trephine in all the cases, except those in which I said that the operation should never be performed. A reference to the foot-note of the tabulated statement will show what is meant. A man must use his judgment. We must take the balance of probabilities, and act on that balance as a general in time of war acts on the information which he has received. We have got to take the risk, and do in obscure cases the best thing possible after weighing all the evidence.

Prof. S. D. Gross has been quoted as giving most valuable rules with reference to trephining. I say with great gratitude that I received instruction from Prof. Gross, in his capacity as teacher, and it is because I have been imbued with his spirit, that I have written what I have. He was ahead of many writers at the time he penned those words. He advocated trephining more earnestly than was the custom at that time.

In regard to wounds of the meningeal artery, Dr. S. W. Gross has given me just now some German statistics showing the increase of life-saving ability from the use of the ligature in such instances.

NEPHRECTOMY:

Its Indications and Contra-indications.

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Of 233 cases of extirpation of the kidney which I have collated from public and private[1] sources of information 129 recovered, and 104, or 44.63 per cent., died. Of the entire number, 111 by the lumbar incision indicate 70 survivals and 41 deaths, the mortality being 36.93 per cent., while of 120 by the ventral incision, 59 recovered, and 61, or 50.83 per cent., perished. In the remaining 2 cases, both of which proved fatal, the nature of the operation is uncertain.

It is thus to be perceived that the fatality of the abdominal operation is greater by 13.90 per cent. than that of the lumbar operation, a difference which several distinguished laparotomists ascribe to the removal of healthy kidneys after a wound of the loin, or for some condition in which they are not diseased. This

[1] Of the 34 unpublished nephrectomies utilized in the preparation of this paper, I have to thank Bardenheuer for 14, Czerny for 6, Martin for 3, Bantock for 3, Savage for 2, Marcus Beck for 2, Elder for 1, Kœnig for 1, Henry Morris for 1, and Thomas Smith for 1. I have also to express my thanks to Knowsley Thornton for a list of 11 nephrectomies, 3 nephrolithotomies, and 4 nephrotomies; to Lawson Tait for a list of 3 nephrectomies and 12 nephrotomies; to Bryant for 13 nephrotomies; to Savage for 2 nephrotomies; to Peruggi for all the nephrectomies done in Italy; to Tscherning for all the nephrectomies performed in Copenhagen; to Barker for all the nephrectomies done at the University College Hospital; to Gaillard Thomas for notes of his 3 cases of nephrectomy; to Hicquet for the termination of his case of nephrectomy for sarcoma; to Pepper for an unpublished nephrolithotomy; to Israel for an unpublished nephrotomy; and to Henry Morris for 1 unpublished nephrolithotomy, 2 exploratory operations for renal calculus, 3 nephrotomies, and for many references to all of these operations. It should be stated that the statistics are brought up to May 15, and that cases reported merely as doing well several days after operation are excluded.

statement is, however, incorrect. For ureteral fistulæ, rupture of the ureter, protrusion through the loin, a painful condition, and excessive mobility, healthy or injured kidneys have been removed in 39 cases. Of these, 19 were by the lumbar incision, with 4 deaths, or a mortality of 26.31 per cent., and 20 by the ventral incision, with 8 deaths, or a mortality of 40 per cent. The greater success of the lumbar operation is also shown by a comparison of the two methods for diseased kidneys. Thus, if we omit the 39 cases just referred to, along with 5 in which the organ or ureter was injured during the extirpation of abdominal tumors, or the kidney was inseparably attached to uterine or retro-peritoneal tumors, 92 lumbar operations indicate 37 deaths, or a mortality of 40.21 per cent., and 95 abdominal operations show 48 deaths, or a mortality of 50.52 per cent. The nature of the disease for which the kidney is extirpated, however, influences the result, since we find that the mortality is less by 25, 16.17, 8.67, and 19.41 per cent., respectively, for suppurative lesions, hydronephrosis, painful floating kidney, and malignant growths, while it is greater by 39.56 per cent. for tubercular kidney, after the lumbar operation. Hence, we may conclude in a general way that the lumbar operation is the safer.

The following table of the causes of death after the lumbar and abdominal methods shows at a glance not only the dangers of nephrectomy, but the comparative frequency of the different causes after each operation:—

Causes of Death.	Abdominal.	Percentage.	Lumbar.	Percentage.
Shock	20	32.79	16	40.00
Peritonitis	13	21.31	1	2.50
Septic peritonitis	8	13.11	0	
Uræmia	4	6.55	2	5.00
Exhaustion	4	6.55	4	10.00
Septicæmia and pyæmia	3	4.91	7	17.50
Anuria	2[1]	3.27	6[2]	15.00
Infarction	2	3.27	0	
Hemorrhage	4	6.55	0	
Secondary hemorrhage	1	1.63	1	2.50
Suppuration of opposite kidney	0	. . .	1	2.50
Vomiting	0	. . .	1	2.50
Convulsions	0	. . .	1	2.50
	61		40[3]	

[1] The opposite kidney was diseased in one.

[2] The opposite kidney was diseased in four.

[3] The cause of death is unknown in one.

From the above comparison it appears that peritonitis, septic peritonitis, pulmonary embolism, primary hemorrhage, and uræmia are more common causes of death after abdominal extirpation of the kidney than after lumbar, and that shock, exhaustion, septicæmia, pyæmia, anuria, secondary hemorrhage, suppuration, convulsions, and vomiting are more frequent causes of death after lumbar nephrectomy than after abdominal. In other words, by selecting the lumbar operation the risks of peritonitis are reduced to a minimum, septic peritonitis is unheard of, as are also pulmonary embolism and primary hemorrhage, which were the causes of upwards of six-tenths of the deaths after the abdominal operation.

Such are the general statements in regard to nephrectomy; but to draw any conclusions as to the real value of this grave operation, it must be considered in connection with the various diseases or lesions for which it has been performed, and compared with other modes of treatment. From an extensive acquaintance with the literature of the subject, I have been long convinced that the kidney has been too frequently removed; and it is for this reason, as well as for the purpose of comparing the relative value of nephrectomy, nephrotomy, nephrolithotomy, nephrorrhaphy, and other surgical measures, and concluding which of these operations should be resorted to in particular cases, that I have undertaken the present inquiry.

1. *Suppurative Lesions.*—For pyonephrosis, pyelitis, and abscess, including one example of abscess after a shot wound, and one of suppuration following traumatic rupture, the kidney has been removed 50 times,[1] with 28 recoveries and 22 deaths, or a mortality of 44 per cent. Of the 40 lumbar operations, 26 recovered and 14, or 35 per cent., succumbed, while of the 9 ventral incisions, 2 recovered and 7, or 77.77 per cent., died. The remaining one case, in which the nature of the operation is un-

[1] The operators were Bardenheuer in 12 cases; Czerny in 7 cases; Tait and Von Bergmann, each in 2 cases; Barwell, Beck, Clementi, Couper, Dandridge, D'Antona, De Vecchi, Dumreicher, Duncan, Elder, Frattina, Israel, Kidd, Lange, Lloyd, Marsh, Ollier, Palmer, Roberts, Savage, Stockwell, Studsgaard, Von Bruns, Von Mandach, Weir, West, and Whitehead, each in 1 case.

known, proved fatal. The cause of death was determined in 20, of which 5 were from shock, 2 from shock and disease of the remaining kidney, 4 from septicæmia, and 1 each from pyæmia, primary hemorrhage, secondary hemorrhage, uræmia, suppression of urine, vomiting, peritonitis, convulsions, and exhaustion. In 1 of the fatal lumbar operations the pleura was wounded, while in two the peritoneal cavity was opened, and one recovered. In 8 cases the kidney had been previously drained by nephrotomy, and 7 recovered.

Incision with drainage of suppurating kidneys has been practised in 72 cases,[1] of which 59 recovered and 13, or 18.02 per cent., died; 67 were by the lumbar incision, with 12 deaths, and 5, in the hands of Lawson Tait, were by the ventral incision, of which 1 perished. Of the 59 survivors, sinuses, or fistulæ persist in 19, or 32 20 per cent.

For calculous pyelitis, nephrectomy has been done 23 times,[2] with 13 recoveries and 10 deaths, or a mortality of 43.47 per cent. Of the 16 operations through the loin, 9 recovered and 7, or 43.75 per cent., perished, while of the 7 ventral operations, 4 recovered and 3, or 42.85 per cent., died. The cause of death was shock in 4, shock and disease of the opposite kidney in 1, anuria in 2, the other kidney being involved in one, and exhaustion, peritonitis, and pyæmia each in 1. Previous nephrotomy had been resorted to in 4, all of which recovered.

Nephrotomy for calculous pyelitis has been resorted to 21 times,[3] with 12 recoveries and 9 deaths, or a mortality of 42.85

[1] The operators were Bryant in 9 cases; Tait in 7 cases; Thornton in 4 cases; Barker, Gardner, Morris, Parker, and Wheelhouse, each in 2 cases; and Angelini, Baker, Bardenheur, Barwell, Beck, Burrell, Callender, Crane, Czerny, Datillo, Elder, French, Goodridge, Gueterbock, Holmes, Israel, Ker, Keys, Krannhals, Kuester, Landau, Lange, Lente, Lister, Lucas, Ollier, Paterson, Puzey, Raffa, Reeves, Roddick, Rosenberger, Savage, Southey, Studsgaard, Van Buren, Von Bergmann, Von Muralt, Weir, West, Williams, and Wright, each in one case.

[2] The operators were Barker, Godlee, and Thornton, each in 2 cases; and Bantock, Barwell, Bottini, Israel, Lange, Macewen, McClelland, Müller, Novaro, Rosenbach, Simon, Thomas Smith, Sonnenberg, Tait, Urginati, Von Bergmann, and Wright, each in one case.

[3] The operators were Bryant in 2 cases, and Anderson, Baker, Beck, Callender, Clark, Couper, Cullingworth, Dawson, Durham, Haward, Hulke, Ingalls, Israel Jones, Mynter, Richardson, Rose, Seymour, and Studsgaard, each in 1 case.

per cent. Of the recoveries a sinus remained in 1, and in 1, a case of Bryant's, a urinary fistula persists at the end of eight years.

It will thus be seen that of 73 nephrectomies for suppurative lesions from all causes, 41 recovered and 32, or 43.97 per cent., died. Of 93 nephrotomies, on the other hand, 71 recovered and 22, or 23.65 per cent., died. It, moreover, appears that of 12 nephrectomies after a previous nephrotomy only 1, or 9.33 per cent., succumbed, while of 61 nephrectomies without preliminary incision and drainage, 31, or 50.81 per cent., perished. In other words, the mortality of removal of the kidney for suppurative conditions is nearly twice as great as after nephrotomy, and a preliminary nephrotomy diminishes the risks of nephrectomy by 41.48 per cent. Hence, we may conclude that a suppurating kidney should never be excised until after the failure of nephrotomy, the only inconvenience of which is that a permanent sinus or urinary fistula remains in 29.57 per cent. of the recoveries, either of which may, however, should it prove a source of discomfort, be remedied by subsequent lumbar extirpation of the shrunken sac, with the risk of only 9.33 per cent. In favor of a preliminary nephrotomy, it is also to be declared that it enables us to determine with almost a certainty the condition of the opposite organ, that is to say, if, after a few days of drainage, pus ceases to appear in the urine, the probability is that the other kidney is sound.

2. *Tubercular Kidney.*—The kidney has been extirpated for so-called strumous disease 20 times,[1] of which 12 recovered, and 8, or 40 per cent., died—3 from exhaustion, along with disease of the opposite kidney in one, and tubercle of the bladder in one, 2 from shock, 2 from suppression of urine, in both of which the remaining organ was similarly affected, and 1 from tubercle of the opposite kidney and of the lungs. Of the 13 operations through the loin 6 recovered, and 7, or 53.84 per cent., succumbed, while of 7 ventral operations 6 recovered, and 1, or

[1] The operators were Thornton in 3 cases, and Baker, Bantock, Beck, D'Antona, Elder, Golding-Bird, Jowers, Lucas, Martin, May, O'Reilly, Peters, Raffa, Tschierning, Von Muralt, Wright, and Wylie, each in 1 case.

14.28 per cent., died. Previous nephrotomy had been resorted to in 8, with 5 recoveries and 3 deaths, or a mortality of 37,5 per cent., whereas primary extirpation was attended with a mortality of 41.66 per cent. In 10 of the recoveries in which the fate of the patient is known, 1 expired in four months from tubercle of the opposite kidney, 1 received no benefit, the bladder and remaining organ being diseased, while 8 were living and well at the expiration, respectively, of two months and twenty days, five months, five months, ten months, twenty-six months, thirty months, three years, and nearly four years.

It will thus be seen that in 7 of the 20 cases there was tubercle of the opposite kidney in 4, of the bladder in 1, and general tuberculosis in 2. Hence, present experience shows that tubercle is limited to a single kidney in only 65 per cent. of all cases, which renders the prognosis far more grave after nephrectomy than after removal of the kidney for suppurative lesions from ordinary causes. In the 8 cases of preliminary nephrotomy relief was not afforded; so that it appears as if previous incision might be wisely refrained from, and the kidney be extirpated in the early stage of tubercular disease, especially as excision may relieve the patient of a source of general infection. In performing the operation we ought, if possible, to ascertain the condition of the opposite organ; and as this may be done by the ventral incision, which is attended with a mortality of 14.28 per cent., as against 53.84 per cent. for the lumbar operation, we may conclude that primary exploration and removal by the abdominal incision is the proper procedure for the early stage of tubercular kidney. In advanced cases, on the other hand, especially if the morbid changes be not limited to the kidney, nephrectomy is clearly unjustifiable, and nephrotomy should be resorted to.

3. *Hydronephrosis.*—There are 21[1] recorded cases of nephrectomy for hydronephrosis, including 5 due to calculi, the case of Heywood Smith being omitted as the result is unknown. Of

[1] The operators were Thornton in 3 cases; Bardenheuer, Czerny, and D'Antona, each in 2 cases; and Billroth, Cullingworth, Day, Goodell, Heath, Le Dentu, Kehrer, Savage, Schede, Spiegelberg, Thiersch, and Thomas, each in 1 case.

these 21, 13 recovered, and 8, or 38.09 per cent., died. Of 17 ventral operations, 10 recovered, and 7, or 41.17 per cent., succumbed, while of 4 lumbar operations, 3 recovered, and 1, or 25 per cent., died. Of the survivors, 2 had been subjected to nephrotomy, and a fistula remained in 1. The cause of death was shock in 3, peritonitis in 2, septic peritonitis in 2, and anuria from disease of the opposite kidney in 1.

Incision with drainage of hydronephrotic kidneys has been practised in 25 cases,[1] of which 21 recovered, and 4, or 16 per cent., perished. Of 14 ventral incisions, 11 recovered, and 3, or 21.42 per cent., died, while of 11 lumbar incisions, 10 recovered, and 1, or 9.09 per cent., died. Of the 20 survivors, urinary fistulæ persist in 11, or 55 per cent. In one of these cases, Mr. Henry Morris writes me that about ten ounces of urine are passed daily into a receiver adapted to the loin, and that the patient suffers neither inconvenience nor discomfort. In a second case, recorded by Taylor, the ruptured sac was stitched to the abdominal wound, and the patient passes urine freely through a glass tube, much to her discomfort. In the cases of Le Dentu and Spiegelberg the trouble was remedied by removal of the kidney.

Of the remaining operations for hydronephrosis, 15 cases of puncture, of which I possess notes, resulted in 12 deaths, 1 cure, and 2 temporary improvements, while of 5 examples of injection with tincture of iodine, 2 were cured, and 3 died. Hence, further efforts in these directions should be abandoned. Of 6 instances of aspiration, 3 were cured, 2 improved, and 1 died of a cause not connected with the operation.

We have thus seen that aspiration is a perfectly safe procedure; that lumbar nephrotomy yields fewer deaths by 15.91 per cent. than does lumbar nephrectomy; and that the latter operation is safer by 16.17 per cent. than is the ventral operation. Hence, in cases of hydronephrosis, lumbar aspiration,

[1] The operators were Czerny in 3 cases; Simon and Weir, each in 2 cases; and Ahlfeld, Baum, Cabot, Kœnig, Le Dentu, Landau, Leisrink, Morris, Pernice, Peters, Savage, Schramm, Spiegelberg, Svensson, Taylor, Tuckwell, Sir Spencer Wells, and Winckel, each in 1 case.

repeated if it be necessary several times, should first be resorted to. In the event of its failure, nephrotomy should then be practised. Should an annoying urinary fistula result, the kidney may be removed through the loin, as a final resource.

4. *Cysts.*—For cysts of various kinds, the kidney has been removed 15 times,[1] with 8 recoveries and 6 deaths, or a mortality of 46.66 per cent. With one exception, all the operations were ventral, and the cause of death was peritonitis in 2 cases, and shock, infarction of the lungs, secondary hemorrhage, septicæmia, and pyæmia, each in 1 case.

Nephrotomy for cysts has been done in 7 cases[2] by the ventral method, all of which recovered, and in Mr. Thornton's case, both kidneys were drained. These results speak for themselves. Nephrotomy should always be tried at the outset, unless it be deemed wise to first resort to the simpler remedy, aspiration. Should nephrectomy unfortunately be decided upon, the same rule should guide us as in nephrectomy for hydronephrosis, since it is probable that all cysts can be removed by the loin after previous drainage.

5. *Painful Floating Kidney.*—Floating kidneys have been extirpated 22 times,[3] with 13 recoveries and 9 deaths, or a mortality of 40.90 per cent. Of 19 ventral operations, 11 were cured, and 8, or 42.10 per cent., succumbed, while of 3 lumbar operations, 2 recovered, and 1, or 33.33 per cent., died. Although not strictly a lumbar operation, Polk's fatal case of extirpation of a floating solitary kidney is included in the latter class, as the incision was extraperitoneal. The cause of death in the 8 fatal cases was peritonitis in 4, uræmia in 2, and septic peritonitis and shock, each in 1 case.

[1] The operators were Bardenheuer, Burgess, Campbell, C aus, Czerny, D'Antona Esmarch, Getz, Keeling, Landi, Leopold, Maclean, Meadows, Ollier, and Walter. The case of Speigelberg is omitted, as the removal was incomplete. It was a ventral operation, and death ensued from shock.

[2] The operators were Tait in 5 cases, and Thornton and Heusner, each in 1 case.

[3] The operators were Martin in 8 cases; Langenbuch in 2 cases; and Billroth, Boothby, Gilmore, Hennig, Jurié, Lane, Martini, Merkel, Navratil, Polk, Smyth, and Stimson, each in 1 case.

Nephrorraphy has been practised 18 times,[1] with 1 death, or a mortality of 5.55 per cent. The operation, however, utterly failed to fix the kidney in 4 of the survivors, and in 3 the success was only partial. Hence, although it has not afforded relief in 43.33 per cent. of all cases, its comparative freedom from danger warrants the conclusion that nephrectomy should never be performed for a movable kidney until mechanical appliances and stitching the organ to the posterior abdominal wall have failed to relieve suffering. In this event it may be removed, as has been successfully done by Gilmore and Jurié; and, although the ventral operation may be easier, especially if there happens to be a long mesonephron, its high rate of mortality from peritonitis should induce the surgeon to select the lumbar incision, through which the organ may almost invariably be readily removed.

6. *Malignant Growths.*—Carcinomatous and sarcomatous kidneys have been extirpated in 49 cases, of which 19 recovered, and 30, or 61.22 per cent., died. Of 37 ventral operations, 13 recovered, and 24, or 64.86 per cent., succumbed, while of 11 lumbar operations, 6 survived, and 5, or 45.45 per cent., perished. Of the survivors, there were recurrence and death in 7, 1 died of an unknown cause, 1 was alive with recurrence, 7 remained well for twenty-eight months and a half on an average, and 3 were living for periods varying from three weeks to two months after operation, whence they are scarcely available for the determination of the final result. In a general way it may, therefore, be said that 61.22 per cent. of all cases die as the direct result of the procedure; that 16.32 per cent. perish subsequently; that 12.24 per cent. appear to make a permanent recovery; and that the lumbar incision is far less dangerous than the ventral. In order, however, that the true value of nephrectomy for malignant disease may be properly estimated, it is necessary that sarcoma and carcinoma be examined separately.

[1] The operators were Hahn in 5 cases; and Agnew, Bassini, Ceccherelli, Dunning, Esmarch, Gardner, Gilmore, Jurié, Kuester, Newman, Svensson, Von Bergmann, and Weir, each in 1 case.

The kidney has been removed for sarcoma 33 times,[1] with 14 recoveries and 19 deaths, or a mortality of 57.57 per cent. Of 26 ventral operations, 11 recovered, and 15, or 57.69 per cent., died, while of 7 lumbar operations, 3 recovered, and 4, or 57.14 per cent., perished. The cause of death was shock in 7, hemorrhage and septic peritonitis, each in 3, septicæmia and exhaustion, each in 2, and pulmonary embolism and tetanus, each in 1. Of the 14 survivors, 6 subsequently died, 1 from an unknown cause, and 5 from metastases at the expiration, respectively, of five months, five months and three weeks, six months, nine months, and eighteen months, while 1 was living with recurrence at the end of four months, and 5 were alive and well at the end, respectively, of seventeen, twenty-two, and twenty-three, and thirty-five months, and five years, the cases being those of Billroth, Lossen, Thornton, Bardenheuer, and Martin. In the remaining 2 cases the history terminates with the mere statement that the patient recovered. In other words, 57.57 per cent. died as the direct result of the operation, while, of the survivors, 42.85 per cent. died subsequently, and 35.71 per cent. remained well for thirty-one months and a half on an average.

The ages of 16 of the subjects ranged between sixteen months and seven years. Of these 16 children, 7 survived, and 9, or 56.25 per cent., perished. Of the 7 survivors, the patient of Kœnig was living with recurrence at the end of four months, while the patients of Bardenheuer, Godlee, Jessop, and Hicquet died of recurrence, respectively, in five, six, nine, and eighteen months. In the remaining 2 there is no further history. In 2 of the deaths, secondary deposits were found in other organs.

[1] The operators were Czerny in 6 cases; Bardenheuer in 3 cases; Kœnig in 2 cases; and Billroth, Bantock, Barker, Godlee, Heath, Hicquet, Hueter, Jessop, Kocher, Little, Lossen, Martin, Meredith, Morris, Ollier, Rawdon, Réczey, Schœnborn, Tait, Thornton, Von Bergmann, and Whitehead, each in 1 case. Langenbuch's case of recovery after the lumbar incision is omitted, as the organ, converted into a cyst, was thrown away, and could not be examined. The fatal cases of Kocher, Israel, and Thiersch are also omitted, as the adhesions were so great that the diseased kidneys were not removed. Trendelenburg also opened the belly, but the extreme vascularity of the tumor prevented him from attempting its removal. The child recovered.

The remaining 17 patients were adults. Of these, 7 recovered, and 10, or 58.82 per cent., died. Of the survivors, 1 died from an undetermined cause; 1 died from metastasis; and 5 were well at the end of thirty-one months and a half on an average. From these data we may conclude that nephrectomy for sarcoma in children should not be repeated, but that in adults it is eminently justifiable, as it apparently cures 29.41 per cent. of the cases. Should the tumor be small, the lumbar incision is the preferable one.

Nephrectomy for carcinoma has been practised in 14 cases,[1] of which 4 recovered, and 10, or 71.42 per cent., perished. All of the 9 abdominal sections, with 1 exception, were fatal, 1 of the 4 lumbar operations died, and 1 died, the nature of the operation being unknown. The cause of death was determined in 9 cases, of which 2 each were due to uræmia, shock, and peritonitis, and 1 each to metastasis, exhaustion, and anuria, the opposite kidney being cystic and contracted. Of the 4 survivors 2 died of secondary growths at the expiration, respectively, of forty-four days and two months, and the remaining 2 were alive at the end, respectively, of two months and thirteen months. Hence, carcinoma should be excluded from the category of cases for which nephrectomy should be done.

In 2 other cases the kidney has been extirpated by the ventral operation for malignant disease. The case of Wolcott, one probably of medullary carcinoma from the age of the patient, who was fifty-eight years old, died of exhaustion, while that of Byford was living at the end of twenty-eight months. As the subject was thirty-six years of age, the disease was probably sarcomatous. Hence, these additional cases of malignant disease confirm our conclusion that nephrectomy is indicated in sarcoma of adults, and contraindicated in carcinoma.

7. *Other Neoplasms.*—Czerny removed an adenomatous kidney of a child eleven months of age by the ventral operation, and death ensued from septic peritonitis. Thomas and Bruntzel

[1] The operators were Adams, Albert, Czerny, Davy, Esmarch, S. W. Gross, Homans, Halstead, Lücke, Martin, Péan, Thomas, Vercelli, and Sir Spencer Wells.

succeeded in saving their patients after laparotomy for fibroma of the capsule of the kidney; and Billroth operated successfully through the loin for papilloma, Peaslee removed a "solid" tumor by laparotomy, but the patient died of peritonitis. For benign tumors, 4 nephrectomies indicate, therefore, 3 recoveries and 1 death, or a mortality of 25 per cent., so that the operation is eminently justifiable.

8. *Calculus.*—The otherwise healthy kidney has been extirpated for calculus by Czerny, Kosinski, and Morris. All of the patients recovered, 2 of the operations having been performed through the loin, and 1 through the belly. Despite these favorable results, no prudent surgeon will sacrifice the organ until all methods of exploration, including incision of the kidney itself, have failed to detect the concretion. When the organ is not dilated by pus or urine, in other words, when its secreting power is not materially damaged, the only operation that is at all justifiable is nephrolithotomy, an operation of which I have a record of 21 cases.[1] Of these, 2, or 9.52 per cent., died—the patient of Cullingworth from the blocking of the opposite ureter by a stone near its vesical orifice, while that of Pepper expired suddenly, in consequence probably of the effects of morphia. Thornton's cases were by the combined ventral and lumbar incision, and all recovered; but under no circumstances should any except the lumbar incision be resorted to, unless there is doubt as to which kidney is involved. In 3 of the cases the stone was removed through an incision in the pelvis of the kidney, and in 2 cases the stones weighed, respectively, nearly an ounce, and five hundred and fifty-six grains. All of the recoveries were prompt and perfect, except in the first case of Morris, who informs me that a sinus still persists, five years subsequently.

When the symptoms point to a renal calculus, the surgeon should make an exploratory operation through the loin, with a view to its detection and removal without a resort to nephrec-

[1] The operators were Thornton in 3 cases; Morris and Beck, each in 2 cases; and Butlin, Bardenheuer, May, Howse, Hill, Symonds, Anderson, Godlee, Elder, Cullingworth, Pepper, Chiene, Rouse, and Tiffany, each in 1 case.

tomy. Of 23 exploratory operations, of which 3 were done by Beck, 2 each by Morris, Lucas, and Annandale, and 1 each by Gunn, Durham, Lente, Golding-Bird, Barker, Barbour, Norton, Anderson, Fowler, Powell, Thornton, Hulke, Bardenheuer, and Lawson, all recovered, the kidney having been actually incised in four. Although a stone was not detected, the procedure was absolutely free from danger, but a calculus was doubtless present in some of the cases, even though it was not found by the fingers and acupuncture. Hence, we may conclude, with Morris, that in calculus of an otherwise healthy kidney, the organ should be incised after the failure of the ordinary methods of exploration, and thoroughly examined with the finger, before it is condemned to removal.

9. *Ureteral Fistulæ.*—For urinary fistulæ communicating with the exterior of the abdomen, the vagina, or the uterus, and due to wounds or injury of the ureter in ovariotomy and hysterectomy, to the use of the forceps in labor, and in one instance to a suicidal attempt, the kidney has been removed 12 times,[1] with 9 recoveries, and 3 deaths, or a mortality of 21.27 per cent. The cause of death was collapse in 2, and uræmia from disease of the opposite kidney in 1. Of the 11 lumbar operations, 3, or 27.27 per cent. succumbed, while the single abdominal operation recovered.

Although it seems a pity to remove a functionally active organ, yet the constant escape of urine in this class of cases is a source of so much discomfort and disgust that it is the only resource. The lumbar incision is, of course, the preferable one.

10. *Injury to the Ureter and Kidney.*—During the removal of a retroperitoneal cystic tumor by laparotomy, Thornton wounded the ureter, and at once extirpated the kidney with success. Bardenheuer met with the same accident during hysterectomy for carcinoma, and removed the kidney through the loin, but the patient died of collapse. Archer lacerated the kidney during ovariotomy, and extirpated it successfully on account of hemor-

[1] The operators were Albert, Billroth, Boeckel, Credé, Czerny, Le Fort, Novaro, Simon, Starck of Dantzic, Starck of London, Thornton, and Zweifel.

rhage. In an example of rupture of the ureter with the formation of a urinary abscess in a child forty-four months of age, Barker removed the kidney through the loin, and the patient recovered. He informs me, however, that at the end of four months the child was dying from general tuberculosis. In these four cases the kidney was perfectly normal, and as its removal was complicated in three by simultaneous severe operations, the slight mortality is simply wonderful. In a fifth case, Rawdon extirpated the kidney through the loin, on account of subcutaneous laceration, and the patient expired on the fortieth day from suppuration of the opposite kidney. In three examples of protrusion of the kidney through a wound in the loin, in two of which it had been incised, Brandt, Marvaud, and Hamilton successfully removed the organ.

Agnew refers to a case in which, after the accidental division of the ureter in ovariotomy, the renal end was secured in the abdominal wound, and a urinary fistula established. The patient died, at the expiration of several months, of severe obstruction of the duct. In removing the uterus for carcinoma, Henry Morris excised half an inch of the ureter, and fixed its renal extremity in the vagina. The formation of a urinary fistula was, however, prevented by the death of the patient.

While Agnew and Morris recommend the establishment of a urinary fistula when the ureter is wounded in removing an abdominal tumor, we consider their proposal as being one of very questionable value, unless, after the formation of the fistula, nephrectomy be performed to rid the patient of a source of great discomfort and annoyance. The fixing of the renal end of the ureter in a wound made in the loin, would certainly lessen the risks of death from shock, which occurred once in the two cases of combined laparotomy for abdominal tumors and nephrectomy. Hence, in this class of injuries, we believe that nephrectomy should not be performed as a primary operation.

Should the kidney be badly lacerated during the removal of abdominal tumors, and the hemorrhage be troublesome, the organ will have to be removed, as was done by Archer. Subcutaneous rupture of the ureter, followed by the formation of a

urinary abscess, demands extirpation of the kidney; but we require more experience to determine the proper course to pursue in subcutaneous laceration of the kidney. Nephrotomy and drainage should probably be tried before the kidney is excised. When the kidney protrudes through a wound in the loin, provided it can be returned, and it is not materially injured, primary nephrectomy should not be performed.

11. *Unclassified Lesions.*—With a view to completing the statistics of nephrectomy, it may be stated that three lumbar operations have been performed by Durham, Bardenheuer, and Langenbuch, of which two recovered. In the case of Durham, which proved fatal from pyæmia, the kidney was painful, but healthy. Bardenheuer's case was supposed to be one of calculus, the patient having suffered from renal colic and hæmaturia; while in that of Langenbuch, performed for supposed sarcoma, the kidney was converted into a sac, and was accidentally thrown away before its true nature could be determined. In two additional operations, done through the belly, Billroth and Sir Spencer Wells removed a sound kidney attached to a uterine growth. Both patients died.

From a careful analysis of all the facts pertaining to the surgery of the kidney contained in this paper, based as it is upon a study of nearly four hundred and fifty cases of different operations, I believe that I am justified in formulating the following propositions for discussion:—

1. That lumbar nephrectomy is a safer operation than abdominal nephrectomy.

2. That primary extirpation of the kidney is indicated, first, in sarcoma of adult subjects; secondly, in benign neoplasms at any age; thirdly, in the early stage of tubercular disease; fourthly, in rupture of the ureter; and, lastly, in ureteral fistula.

3. That nephrectomy should not be resorted to until after the failure of other measures, first, in subcutaneous laceration of the kidney; secondly, in protrusion of the kidney through a wound in the loin; thirdly, in recent wounds of the kidney or of the ureter, inflicted in the performance of ovariotomy, hysterectomy, or other operations; fourthly, in suppurative lesions; fifthly, in

hydronephrosis and cysts; sixthly, in calculus of an otherwise healthy kidney; and, finally, in painful floating kidney.

4. That nephrectomy is absolutely contraindicated, first, in sarcoma of children; secondly, in carcinoma at any age, unless, perhaps, the disease can be diagnosticated and removed at an early stage; and, thirdly, in the advanced period of tubercular disease.

NEPHROLITHOTOMY.

By L. McLANE TIFFANY, M.D.,
PROFESSOR OF SURGERY IN THE UNIVERSITY OF MARYLAND.

I was asked, in February of this year, by Dr. Frank Donaldson, Jr., of Baltimore, to see with him H. J., a patient, in the pelvis of whose right kidney Dr. Donaldson had diagnosticated trouble, for the relief of which surgical treatment was judged necessary. The patient was a male, aged twenty-six years, married, the father of one child, pale, emaciated, and gave the following history: at the age of seventeen years he began to experience pain in the right loin, paroxysmal in character and not very intense. The attacks of pain gradually increased in severity and at shorter intervals; he was, however, able to finish his apprenticeship, that of tinner. About six years ago he noticed that his urine was lightish in color and deposited a white sediment, as he expressed it. At that time exercise did not bring on an attack of suffering, neither did he observe blood in his urine. On July 4, 1884, he was seized with an attack of pain extremely severe on the right side just below the ribs; pain has continued almost constantly ever since, remissions being rare; during the night pain has been more severe than during the day. Since last July, the patient under consideration has taken much morphine; without it, he says, he could not live.

Physical examination showed tenderness on percussion, and on pressure acute pain over the front of the belly about the region of the kidney; no tumor was detected, although searched for.

The outer portion of the right external oblique was rigid, and the lumbar muscles were more rigid than natural; percussion gave a sound somewhat more dull than over the corresponding region of the left side. The patient moves slowly, holding the trunk stiffly for quick motion, and any exertion causes much pain.

Urine was passed in normal amount, and contained much pus; casts were not observed, although searched for; there was no history of blood or gravel having been passed at any time. Micturition not unduly frequent or painful, and in full stream.

These symptoms justified exploration of the kidney, so on February 21st, the patient being anæsthetized with ether, I exposed the kidney by an incision carried half an inch below and parallel to the last rib. The quadratus lumborum muscle was divided, as also the outer edge of the erector spinæ. The perirenal and subperitoneal fat being divided, the kidney was watched and seen to rise and fall regularly with respiration, thus excluding perirenal inflammation to any marked degree. To the touch the gland seemed more hard than normal, and a needle passed into the kidney did not touch stone; on passing the needle again in, it was directed more forwards than the first time; I at once struck a stone. The kidney was then freely opened lengthwise with the hot blade of Paquelin's cautery, and the finger passed in to explore. The kidney pelvis was filled by stone so closely that without undue force I was not able to pass any forceps so as to grasp the calculus. In attempting so to do, I broke off a piece of stone and saw that I had to do with a phosphatic concretion.

I then directed an assistant to press with the flat of his hand upon the patient's belly so as to force the kidney towards my incision, while with a Volkmann's sharp spoon and forceps I broke up and removed the stone. The spoon acted charmingly, cutting a hole in the centre of the mass with rapidity. The débris was washed away by means of a syringe. The fragments which were collected weighed 556 grains—phosphate of lime. Hemorrhage was insignificant and ceased under hot water irrigation. A large drainage-tube was passed into the pelvis of the kidney and an oakum and iodoform dressing applied. At the (12 M.) conclusion of the operation the pulse was 156, respiration 18, temperature 101.4°; at 11 P. M., the pulse 132, temperature 101°, respiration 18. Forty minutes after the operation the patient passed water and complained of its burning him.

Convalescence calls for no special comment. All went well;

the patient sat up out of bed and had full diet in a week and left the hospital three weeks and four days after operation; the wound being reduced to a healthy granulating surface, level with the skin, through which no urine came; it has since healed perfectly, and gives no discomfort.

Two noticeable facts became apparent as healing went on: 1st. That during the early days after operation when the urine from the wounded kidney came through the back, the urine from the bladder was clear and almost quite free from pus, as however the incision closed, pus again appeared in the urine, indicating that the kidney not opened was probably healthy.

In the second place, it was found possible to inject water into the drainage-tube and so through the ureter into the bladder. Pus was still to be found in the patient's urine when he left the hospital, but of course in greatly diminished quantity.

While the term nephrolithotomy is not a new one, yet it has been revived, defined, put upon a good surgical basis, and appended to a successful case by Henry Morris (vol. xiv. *Clinical Soc. Trans.*), to whom is due much credit. It is used to indicate the removal of a stone or stones from an otherwise healthy kidney, as distinguished from such operation as may be necessary to remove a stone or stones from a kidney dilated by pus or urine, or where the stone may have escaped by ulceration into the perinephritic tissues, or where a sinus opening externally conducts the operator to the stone, or indeed where the kidney may present evidence of much disease.

A lumbar swelling which fluctuates is opened, discharges pus and permits the operator's finger passed through the incision to touch a stone free in the abscess cavity, is a very different procedure from that for removal of a small stone from the kidney cortex which has not set up suppuration, yet I venture to hope that at no distant period the one will be operated for as certainly as the other. The matter is an affair of diagnosis, to obtain which it is most fitting to question the experience of others.

Four-fifths probably of individuals hitherto subjected to nephrolithotomy have been young, between 19 and 30 years inclusive, but it is fair to suppose that this experience will be

changed somewhat as the necessity for the operation becomes more widely recognized, since all the patients suffered for years before the stone was removed; a calculus is removed from the urinary bladder when discovered, and I would apply to the kidney pelvis a similar treatment. The male sex largely predominates, in this respect differing not at all from that which one would expect, since calculus of the bladder in the male is so much more often met with than in the female.

I find no occupation apparently particularly liable; statistics, however, are not full enough to warrant any conclusion. From a limited number of observations it would appear as though the left kidney was the more often affected, although the excess is small. The theory of hereditary predisposition is not supported. The symptom which is most prominent throughout, and the one for which the physician is consulted is pain. Pain was complained of for a long time, thus a male aged 50 suffered during 24 years; a male aged 24 suffered since schoolboy days; a female aged 19 since 8 years of age; a male of 19 since 7 years of age; a male of 19 for 8 or 10 years; a female of 23 during the past 7 years, and this is the history with all. With all, the pain for a long time, if not always, is paroxysmal, recurring at first rarely; subsequently, however, the intervals become shorter and shorter, until finally constant suffering may be experienced. The acuteness of the attack is apt to be in direct ratio with its frequent recurrence. This is not an unvarying rule and the intermissions are occasionally long, in one case four and in another ten years of rest being enjoyed. Sooner or later there may occur, however, a marked exacerbation in all symptoms, which I am inclined to attribute to a dislodgement of the stone from its fixed position, thus permitting movement in the kidney pelvis, with injury to the mucous membrane.

Pain is described as follows by different reporters:—

At varying intervals from a day to a week; during the past four years pain has nearly left him, now he has a dull pain on exertion.

Pain in loin shooting to testicle, which is retracted; pain and tenderness on deep pressure in loin.

Shooting pain in right lumbar region, in groin and loin. No tenderness or resistance; this appeared by the time of operation.

Neuralgia of testicle daily and for several hours; during an attack the patient writhed with agony, the testicle was drawn into the canal and could not be touched. An attack was usually preceded by pain deep in the belly, also by lumbar pain.

Pain in loin, side of belly, testicle, inside of thigh. During four years, attacks occurred every six months, then constant pain for two years, and finally during ten months the patient has remained in bed.

Great pain in the loin down the ureter to testis and thigh, followed by hæmaturia. Pain has been observed to shoot down the thigh as far as the knee. Atrophy of the testicle succeeded the pain in one instance. Exertion, in all cases that I have noted, has given rise to pain, and especially such motion as throws the muscles of the loin into action. Sudden active movement induces much suffering in an advanced stage of the malady. The lumbar muscles are occasionally quite strongly contracted and rigid over the affected kidney, and the outer portion of the external oblique may be likewise contracted; under anæsthesia this symptom will disappear. A lumbar swelling and dulness have been in one instance observed to disappear under anæsthesia; doubtless muscular rigidity had much to do with their presence. Frequency of micturition has been noticed several times, particularly after a paroxysm. Symptoms referable to the urine are not very clearly defined, since during the years of suffering the urine will probably have indulged itself with various changes at different times; the urine is thus described in various cases:—

Sp. gr. 1010, neutral, contains blood and triple phosphates; later, urine from the same patient contains albumen and pus, with sp. gr. 1024.

Sp. gr. 1020, acid, trace of pus.

Sp. gr. 1020, in copious quantity during the twenty-four hours; never any pus or blood.

Sp. gr. 1020, alkaline, pus, no blood or casts; a few days later blood appeared and the urine became acid.

Probably a noticeable fact from a diagnostic standpoint is rapid change of urine from alkaline to acid, or the reverse, without apparent cause, dietary or what not. The constituents of the urine may be thought of from a similar standpoint. Blood will probably be found in the urine according as much or little exercise has been indulged in. In one instance it is thus described—the urine is dark with blood after exercise, rest cleared it up. In another instance—blood in quantity has been passed lately after exercise, enough to obstruct the urethra by coagula; after a paroxysm, blood is apt to be present in the urine. Sand and gravel have both been exceptionally met with. Casts of the urinary tubules are observed very rarely, probably because they are hidden by other constituents of the urine. But the indications of stone already noted do not suffice for a diagnosis any more certainly than calculus of the bladder is accepted as present without being touched, so the kidney must be exposed and sounded. It goes without saying that the incision to expose the kidney is to be utilized for extraction if a calculus be found. The incision which is as convenient as any other, is one parallel to the last rib, and about half an inch below it; or the usual colotomy incision will do, and the kidney sought for posterior to the colon. The quadratus lumborum is to be divided and erector spinæ if necessary, since an empty colon moves toward the spine, carrying the peritoneum, and both should be avoided. A thin fascia has been recognized in the perirenal fat, but this may be disregarded, for as soon as this fat is exposed the finger can feel the gland, and it may at once be cut down upon. The length of the various cuts will depend upon the exigencies in each case. The rise and fall of the kidney with the respiratory movements have been observed in more than one instance. Examination of the exposed kidney by touch is to be made from in front, as strongly urged by Howse; the gland is thus pressed against the psoas muscle, which offers resistance, and enables the investigating finger to appreciate changes in density, etc. I think it inexpedient to explore widely in the perirenal tissue, for the kidney will probably be opened, and a split kidney will cicatrize more comfort-

ably with its connection to surrounding parts undisturbed, than not. Failing to recognize a stone by touch, a needle may be passed into the kidney, and the pelvis explored. This is the usual method. I am rather of the opinion, however, that it is needless, and that the kidney which justifies exposure also justifies cutting open and exploration of its pelvis. Acupuncture holes are apt to bleed somewhat for a moment or two. The kidney is to be opened by a longitudinal cut, which bleeds very sharply, but yields to pressure promptly, and probably also to hot-water irrigation. If Paquelin's cautery is used, there is no bleeding. It is interesting to learn that in one case, that reported by Anderson, the stone had two projections like the fangs of a tooth, between which was engaged a piece of renal substance; in extracting, this piece was torn away bodily, yet no harm resulted, and the wound healed promptly. It may be inferred from the foregoing that the kidney is to be treated as other glandular organs of the body, and will heal as promptly. The finger is the best instrument with which to explore the kidney pelvis, as well as the secreting substance from within.

The smallest stone that I have noted is that of Howse, oxalate of lime 26 grains; the largest is the one now reported and shown; the next largest is that of May, crystalline phosphate of lime with nuclei of oxalate, 473 grains. Calculi hitherto extracted are largely made up of lime oxalate and phosphate. The mortality following this operation is small, probably five per cent. or ten per cent. at most, even now in its very infancy, a mortality which may confidently be expected to decrease as we gain experience. It will probably take its place among the most successful of major operations.

Will the kidney, the subject of nephrolithotomy, return to a healthy condition? Time must reply to this query; at present no answer can be given; a sufficient period has not elapsed since Morris's first operation, 1880. On general principles we should expect a return to health after removal of a foreign body, but we know that the age of the patient has much to do with permanent and complete recovery after removal of stone from the bladder, so I am inclined to think that the same may hold

good in regard to the kidney pelvis. May's patient, three months after operation, passed urine sp. gr. 1020, alkaline, containing mucus, pus, and triple phosphates. The possibility of a recurrent attack suggests itself, but we are without data on the subject.

I have mentioned that I was able to inject my patient's bladder with water through the ureter. This has been to me a fruitful theme.

Exploration and catheterization of the ureter from the bladder in the female has been attempted not over successfully, but it could be done from the kidney extremity in either sex. The injection of colored water through the ureter, a catheter having been passed into the bladder through the urethra, would demonstrate its patency in case of pressure by a tumor or calculus; or the latter might be touched by a slender flexible bougie, metal tipped, and extracted. There is still another way in which this suggestion might be utilized, when a nephrotomy has been done and the question of removing the diseased kidney arises, by washing out the ureter, and then plugging it, the urine from the other kidney could be collected and its healthiness investigated, for wherever a nephrectomy is under consideration the fitness of the remaining kidney for the extra work to be thrown upon it will come up for discussion. I am inclined to institute a comparison between the reservoirs at either end of the ureter, when, in addition to the normal fluid contents, a stone is present. In both, certain symptoms occur which we should recognize; in both, we should sound, find the stone, and extract, and I am strongly inclined to believe that in nephrolithotomy, as in lithotomy, the age of the patient, as already stated, has much to do with the outcome. How important then is it to diagnosticate and operate early.

There remain for consideration a certain set of cases, in number not a few, which cases have been diagnosticated as fit subjects for nephrolithotomy by not only competent, but by eminent surgeons, and yet no stone has been found after the kidney was exposed, and also, after the kidney was cut open

and explored by the finger; for instance (*Lancet*, June 2, 1883, p. 948):—

C. S., aged 29 years, married, female, dated her illness from an attack ten years ago, characterized by sudden and excruciating pain in right loin, accompanied by vomiting.

Shortly after this her urine smelt badly, and contained a yellow deposit. She came under the surgeon's care February, 1881; chief symptoms were lumbar pain, increased by exertion, fetid urine, and frequent micturition. Under ether, nothing was found wrong with the bladder, uterus, or rectum. There was no lumbar swelling. Urine acid, and contained pus. January, 1882, on examining the right loin something was felt to slip under the fingers, she at the same time complaining of a sharp, stabbing pain.

On admission she complained of severe pains in right loin, which extended from groin into the labium and down back of thigh; they were increased by exertion, and prevented her from walking any considerable distance. Urine alkaline faintly; sp. gr. 1014; gave reaction to guaiacum blood test. Microscope showed triple phosphates, mucus, pus, shrivelled blood-corpuscles. She got thinner, and became slowly worse.

December 25, 1882, kidney exposed; it was observed to rise and fall in respiration. No resistance could be felt in it. It was punctured in three places with an acupressure needle, a gritty sensation attending the last puncture. The kidney was cut open and the pelvis explored with the finger, but nothing was found. Hemorrhage small, and ceased spontaneously very soon.

The wound was closed, and recovery followed. March 8, 1883, patient was going about her work without pain, but urine continues rather unpleasant. The operator (Lloyd) says: "Whatever her disease is, I feel sure it is situated somewhere between the bladder and the kidney substance, and is out of reach of surgical investigation."

I cannot avoid thinking that the territory between the kidney and bladder is not out of reach of surgical investigation, and that if the methods of exploration already noted in this paper had been employed, something might have been learned concerning the "dark continent." I do not accept the diagnosis of the reporter.

Here is another case (*Bull. de Thérap.*, 1881, 101, p. 343):—

M. X., aged 55 years, suffered from several attacks of left nephritic colic, especially during the past three years, which were followed by the passage of uric acid sand, extremely fine; no piece of gravel had ever been passed. For more than six months continuous pain, with a sense of impending colic, rendered it impossible for him to continue his occupation. Finally, the patient was obliged to remain in bed, or else to occupy an extension chair; walking induced much suffering. The left loin appeared larger and more rounded than usual. Percussion here awakened suffering, as did also pressure. No febrile phenomena existed.

The kidney was exposed by incision; digital examination revealed nothing; acupuncture practised four or five times; a grating sensation, attributed to small sand; later a stone was believed to be struck. With Paquelin's cautery, an incision, six or seven centimetres long, was made into the kidney, but no stone finally found. The wound healed, and complete cure resulted.

The incision through the fibrous capsule, dislodgement of small sand from kidney substance, and revulsive action of cautery are believed to have contributed to a cure; but the operator gives the most credit to free division of the kidney capsule, and with this opinion I concur. The irritation set up by the presence of sand in the kidney substance, or by adjacent inflammation in the pelvis of the kidney, is sufficient to determine a congestion and swelling of the gland, which will, of course, be resisted by the fibrous capsule, hence pain results, and if the pelvis is inflamed, pus, etc., will be in the urine. A similar train of symptoms is seen when the testicle becomes painful and tense from inflammation in an adjacent epididymis. In both cases similar treatment brings relief, namely, division of the fibrous capsule.

With our present knowledge, then, it is apparent that the symptoms of kidney stone may be so closely imitated as to deceive any one, but while the diagnosis is in doubt the treatment is not; a free incision is demanded.

To the comparison already made between bladder and kidney pelvis, I would add another, that both may require free incision,

drainage, and rest, to bring about a cure of chronic inflammation, thus still further applying a common rule to different parts of the urinary apparatus, and so simplifying surgery.

DISCUSSION.

Dr. S. W. Gross, of Philadelphia, Pennsylvania.

I congratulate Dr. Tiffany upon the successful termination of his case, the more particularly because he had removed from an otherwise healthy kidney the largest calculus that has as yet been recorded, the only one approaching it being the concretion, weighing nearly one ounce, excised by Mr. Bennett May.

As Dr. Tiffany has stated, the operation is a comparatively safe one. Thus, of 21 cases that I have collated, 2, or 9.52 per cent., died—the patient of Cullingworth from the blocking of the opposite ureter by a stone, and that of Mr. Pepper, as he writes me, from the effects of morphia. In Mr. Thornton's 3 cases, the operation was by the combined lumbar and ventral incision, while the remainder were by the lumbar incision. In the cases of Butlin, Anderson, and Cullingworth, the stone was removed through an incision in the pelvis of the kidney.

I also agree with the author of the paper in regarding the diagnosis of renal calculus as being far from easy. Intermittent attacks of renal colic, with tenderness on pressure, the presence of blood, and it may be of a little pus, in the urine, with frequency of micturition, are, in the absence of vesical and prostatic lesions, the best diagnostic signs; but they may be present in cases of highly acid urine, and in the early stage of tubercular kidney. The group of symptoms which I have just mentioned being present, the surgeon is certainly warranted, after the failure of ordinary remedies to relieve them, in cutting down upon the kidney through the loin, with the view to its exploration with the fingers, a needle, and, if needed, by an incision of the kidney substance itself, through which the finger can be carried into the calices and pelvis of the organ.

And this brings me to the third conclusion, that incision down to and exploration of the kidney are eminently proper, in which I

entirely agree with Dr. Tiffany. Thus, of 22 exploratory operations which I have collated, all recovered, and the organ was incised in 3. Should incision in every case of failure of the ordinary methods of exploration to detect a stone be the practice of the future, the risks will doubtless be added to ; but I believe that Mr. Morris is right in recommending its adoption. It certainly should be resorted to before the kidney is sacrificed by extirpation.

Dr. J. W. S. Gouley, of New York, N. Y.

I have very little to say except to congratulate the author of the paper on his success in removing such a large calculus from the pelvis of the kidney and also on the successful closure of the wound. In the *Transactions of the New York State Medical Association* for 1884, Dr. W. W. Seymour, reports a case of nephrolithotomy which has proved successful, and in which there were some difficulties in diagnosis, the stone being much smaller, however, than the one just reported. There is still, in Dr. Seymour's case, a fistulous opening in the lumbar region. The exploring needle was used as a director and the opening made with a bistoury. The hemorrhage soon ceased, and the stone was successfully removed after breaking it up. I think that where the stone is of any considerable size, it is not safe to extract it entire. Therefore lithoclasty combined with the operation will be more successful.

Dr. Tiffany mentions the fact that the larger part of the calculus was made up of phosphate of lime. Last year there was considerable discussion on the subject of prostatic calculus and calculus in the prostatic portion of the urethra. A case was presented as one of prostatic concretion, and the ground taken by the author of the paper was that the stone being made up of phosphate of lime, it must be prostatic. I do not think it follows. Here we have a stone from the pelvis of the kidney, the greater part of which is phosphate of lime, and this I think makes good my point of last year.

I think that incision through the substance of the kidney on its outer edge is better than an incision through the pelvis. There is I think less liability to the formation of a permanent fistula, the incision can be larger, and instruments of a larger size may be employed. Another point which has interested me is the use of the thermo-cautery, which is a great improvement in the operation. I should certainly employ Paquelin's cautery, if I were obliged to perform this operation.

Dr. Theodore McGraw, of Detroit, Michigan.

I have just one point to make. We have very little stone in Michigan, either vesical or renal, but I can illustrate one point in the pathology. The vesical stone that we have in Michigan is almost always the result of obstruction. I think that Dr. Gunn can substantiate this statement that it comes from obstructive conditions. The little experience that I have had with stone of the kidney is that it comes from obstruction. The only experience that I have had with it has been on the post-mortem table. Where the ureter was pervious, I have never found small calculi in the kidney; where there has been inflammation of the bladder and ureter with thickening of the ureter, we are liable to have calculi in the kidney. Some time ago I made a post-mortem on a boy of sixteen who had been passing pus in the urine with a deficient amount of urine. There was suppuration in both kidneys, and both ureters were nearly closed. There was a small phosphatic calculus also found. It is, therefore, probable that calculi in the kidney are secondary, not primary.

Dr. P. S. Conner, of Cincinnati, Ohio.

I wish only to congratulate Dr. Tiffany upon the exceedingly successful result of the operation, and to say that his case, in connection with those before reported, makes it more encouraging for us to interfere actively in suspicious cases. Many of these cases present conditions in which the diagnosis is exceedingly difficult. We are encouraged in cutting down and exploring the kidneys, knowing that if we find a stone we shall be successful in relieving the patient, while if we fail no serious damage will have been done the patient.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

I feel that we should congratulate both the gentlemen on their admirable papers. There is one point on which I should like to hear a fuller discussion, that is as to the advisability of cutting through the kidney structure or through the pelvis of the kidney. I do not know that that point was discussed in either paper. What statistics are there to show which is the preferable mode of reaching the cavity? In one case the structure of the kidney is divided. If we can reach the pelvis of the kidney equally well and get as good results by cutting through the fibrous structure of the pelvis, it would probably be the better plan. Experience is what we want.

Dr. Gross. The case mentioned by Dr. Gouley cannot properly be called one of nephrolithotomy, as Dr. Seymour, the operator, distinctly states that the operation was done for calculous pyelitis.

Dr. Tiffany. The case reported by Dr. Gouley as one of nephrolithotomy for calculous pyelitis, would hardly come under the definition of nepholithotomy, according to Morris. It would be opening the kidney for abscess, containing a calculus. A stone forming in the kidney will undoubtedly give rise to suppuration, and pus is usually found in the urine unless dammed up for some reason. In such a case the operation for the removal of the stone may be termed nephrolithotomy. In the case I reported, there was much pus in the urine, but the kidney structure was tightly in contact with the stone and the irregularities of the stone corresponded with the irregularities of the kidneys. It was a mammillated stone.

In regard to the incision. The first case, that of Morris, where a fistula still persists, my recollection is that the incision was made through the secreting substance. Where the pelvis has been opened the wound has always healed. So in seventeen or eighteen cases in which the kidney structure has been opened. In the case that I have reported, I made my incision through the gland structure by preference. The incision usually made exposes the gland structure of the kidney. None of us hesitate to operate on the parotid gland, but all dislike to cut Steno's duct. I think it better to open the kidney structure. The place where the kidney is exposed is the place where I would open it. I think it better to open the kidney where exposed than to disturb the connective tissue and run the risk of having perirenal inflammation.

Dr. Gouley. I should like to say one word more. Both Dr. Gross and the essayist say that this case which I have mentioned, and which I know to be a case of nephrolithotomy, was not a case of nephrolithotomy, and wish to exclude it. If they do that, they will have to exclude every case of stone in the bladder in which the bladder is filled with pus and urine; they will have to exclude all such as cases of lithotomy. Was there ever a case of kidney stone without pyelitis?

Dr. Tiffany has said that the kidney was opened for abscess. It was not opened for abscess. It was a case of pyonephrosis caused

by a stone. The stone was not caused by the pyelitis. Therefore I say that it is a legitimate case of nephrolithotomy, no matter whether the stone filled the pelvis of the kidney, or whether it was surrounded with more or less pus and urine. I do not think that the case should be excluded.

In regard to Dr. Prewitt's question in reference to incision through the pelvis of the kidney I have seen one case in which it was opened, but this was not for the extraction of a stone. I believe that it should not be opened on anatomical grounds. It is better to cut through the convex portion, or the outer border of the kidney. I do not think that it is wise to disturb the connective-tissue in the lumbar region. In the case referred to the pelvis was opened because it was greatly distended.

Another reason for favoring incision through the gland structure is that the stone is reached, as it were, from the base of the cone, the apex being the ureter. The stone can, therefore, be extracted with more ease.

Dr. Gross. In reference to the question of Dr. Prewitt, there have been eighteen cases in which the renal substance has been incised. In three there was quite abundant hemorrhage, which was stopped by plugging the wound with a sponge for less than a minute, so that the risks from hemorrhage are not great. The only danger from opening the pelvis is the formation of a urinary fistula. In three cases, those of Anderson, Cullingworth, and Butlin, the pelvis was incised. I think that the rule may be established to remove the stone from the most accessible point, whether that be the parenchyma or the pelvis.

The operation of nephrolithotomy is the removal of a stone from an otherwise healthy kidney, which is entirely different from removing a calculus from a suppurating or hydronephrotic kidney. Hence, the case referred to by Dr. Gouley must be excluded. In many cases of renal calculi the pelvis is not distended by pus or urine. He speaks from a theoretical knowledge, who says that stone cannot be present without pus in the bladder, and that in every case of stone in the bladder the urine is loaded with mucus and pus.

The President. Dr. Tiffany will close the discussion.

Dr. Tiffany. I have nothing further to say. Perhaps, Dr. Gouley would like to take my place.

Dr. Gouley. I should be glad to do so. I am sorry that Dr. Gross misunderstood me in regard to the condition of the bladder. I did not intend to say that every bladder which contains a stone was filled with pus and urine. The bladder generally contains urine, and it often contains pus. I persist in saying that whenever there is a stone in the pelvis of the kidney, that that stone, acting as a foreign body, must cause a greater or less degree of pyelitis. There is in a large majority of the cases more or less pyelitis. There may be pyonephrosis. There may be a large quantity of urine retained in the pelvis of the kidney from obstruction. Dr. McGraw has referred to obstruction of the ureters. We do see this sometimes in kidney stones. I say on that ground, this case should not be excluded. It happens to be a case of pyonephrosis. I think that I comprehend the subject. I think I know what nephrolithotomy means. This is not a good term, we should have a better one. I wish Dr. Gross or Dr. Tiffany would give us a better one. Cases of kidney stones must show signs of pyelitis. The stones would not become very large if they failed to produce pyelitis. A proof of this is found in Dr. Tiffany's case, in which the stone is made up largely of phosphate of lime, and Dr. Gross knows, as well as we all do, that chronic inflammation of this mucous membrane leads to the formation and deposit of phosphate of lime around a nucleus. That fact has been well established. I do not take one retrograde step from my original position, that in most of these cases there is more or less pyelitis, as there is more or less cystitis in stone in the bladder.

Dr. Gross. In several cases the stone was in the secreting substance of the kidney. In Butlin's case the urine was repeatedly examined without pus being found. I understood you to say that there was pus in the urine in every case of nephrolithotomy.

Dr. Gouley. I said stone in the kidney.

Dr. Gross. It might occur to a person that there are several forms of pyelitis. Pyelitis means inflammation of the pelvis of the kidney. There may be several forms of pyelitis. In some of these cases of extraction of a stone from the pelvis of the kidney, pyelitis of a suppurative nature was not present. Hence the practical fact is better than theory.

Dr. Gouley. There are exceptions to every rule. There are exceptional cases where I will grant there is pyelitis which is not a suppurative pyelitis.

THE HEALING OF ARTERIES AFTER LIGATURE.

By J. COLLINS WARREN, M.D.,
ASSISTANT PROFESSOR OF SURGERY IN HARVARD UNIVERSITY,
BOSTON, MASSACHUSETTS.

Although the introduction of the ligature is commonly ascribed to Paré, there is sufficient evidence to show that it was employed by surgeons in the earliest historic times. No mention is made of the ligature by Hippocrates, but the ancients not only used styptics and the actual cautery, but also ligature and torsion. It is highly probable that the Alexandrians were familiar with the use of the ligature three centuries before the Christian era, for Celsus (born 30 B. C.), speaks of it as a well-known fact, and recommends its use. Rufus of Ephesus, a century later, made following mention of torsion. "*Vas immissa volsella extendemus et moderati circumflectemus: at ubi ne sic quidem cessaverit* [*hæmorrhagia*] *vinculo constringemus.*" Archigenes and Galen both mention tying vessels for the purpose of stopping hemorrhage. The name of Antyllus also bears testimony to the skill of Roman surgeons; and in the museum at Naples, there may be seen a forceps, with sliding attachment, evidently intended to use with the ligature. We find mention of it again in the writings of Paulus of Ægina, in the seventh century, whose teachings were still preserved by the Italians in the sixteenth century.[1] In Lanfranchi's Surgery (1295) occurs the following passage: "*Oportet te nunc aut venam ligare et ipsam de loco extrahere, et caput venæ vel arteriæ contorquere, aut ferro candente sanguinem sistere.*" It is uncertain, however, whether ligatures were employed on large vessels before Paré's time. To this great surgeon is due the credit not only of fully

[1] Hæser Geschichte der Medicin, vol. i.

appreciating the value of this mode of hemostasis, but of making it a universally applicable method. At this period, the middle of the sixteenth century, the imperfect knowledge of the anatomy and physiology of the circulation, prevented a due appreciation of the advantages of the ligature. And even Guillemeau, who was the champion of his friend and teacher, confined the use of the ligature to primary amputations. Although Wiseman, in England, Fabricius Hildanus, in Germany, Fallopius, and others favored the ligature, these were but isolated examples, and at the opening of the eighteenth century the actual cautery was still the customary method of arresting hemorrhage at the Hôtel Dieu. The contrast between the two methods, at that time, was not indeed as great as it would seem to-day. A glance at Paré's plates shows the forceps an instrument of rude pattern and clumsy make; no attempt was made to isolate the vessel, veins, nerves, and arteries being included in one knot. No wonder that surgeons had a "horrid apprehension of compressing the nerves" and that Petit[1] with whom modern investigation on the healing of arteries may be said to have begun, actually proposed compression as a substitute for the ligature. It was he who first called attention to the agency of the thrombus in checking bleeding, the blood around the end of the vessel being termed the *couvercle*, and that found within the lumen the *bouchon*. The retraction and contraction of the vessel were soon recognized by Morand, who also called attention to the rupture of the inner walls by the ligature, Pouteau of Lyons thought that the swelling of the tissues surrounding the mouth of the vessel was as important a factor as anything else, and devised what has since been called the mediate ligature. But this was a return to obsolete methods, and a protest was soon raised against it.

Hunter gave a great impetus to the study of the process of repair in arteries, through his views on healing by first intention. He first enunciated a theory which has led to so much discussion since, the organization of the thrombus and its vascu-

[1] Jean Louis Petit. *Dissertation sur la manière d'arrêter le sang dans les hémorrhagies, Memoir de l'Academie Royale des Sciences.* (73.)

larization, which he likened to the changes seen in the embryo of the chick, but he also believed in a direct adhesion of the walls. It was due to him also, that the subsequent establishment of a collateral circulation was recognized. To Jones,[1] however, has been pretty generally accorded the credit of producing the classical work upon this subject. By a large and varied series of experiments on animals, he was able to give a complete account of the macroscopical appearances following injuries to arteries which in the main holds good to-day. Permanent occlusion, he says, is affected by the inflammation of the walls, the vasa vasorum pouring out lymph, which, according to the theory of that day, became organized, that is, was endowed with an independent power of forming tissue. Jones's views were generally accepted, but as he differed essentially from Hunter, as to the role played by the internal thrombus, this structure became now the chief subject of discussion, one party maintaining that it was organized and formed the cicatricial tissue, the other believing that this function was performed by the walls of the vessel which were supposed to be united by an adhesive inflammation. With the rise and progress of histological research the question of the organization of the thrombus was subjected to new tests. Indeed a study of the question of repair in arteries is a review of the history of the prevailing theories on cell action during the succeeding decades of the present century. We can trace here the work of Schwann and Henle, of Virchow and Rokitansky, of His, Recklinghausen and Cohnheim.

The theory of the organization of the thrombus held its own for nearly half a century, and the name in default of a convenient and appropriate substitute has been preserved to the present day. The organizing power of plastic lymph had to give way to new views on cell action. The discoveries of Recklinghausen and Cohnheim on the action of the wandering cells and the white blood corpuscles brought about further modifications in the

[1] A treatise on the process employed by nature in suppressing the hemorrhage from divided and punctured arteries and in the use of ligature. J. F. D. Jones, M.D., London, 1805.

theory of the process of repair. With new contributions by His to our knowledge of the position in the histological family of the endothelial cell there came a reaction of opinion as to the character of the cell growth which substituted itself for the clot, and closed the open end of the artery ; and at the present time there is almost a unanimity of opinion as to the activity of the cells of the intima both in endarteritis and the repair of arteries after ligature. A more detailed statement of the history of this question has already appeared in the second volume of our Transactions from the able pen of our Fellow Dr. N. Senn.

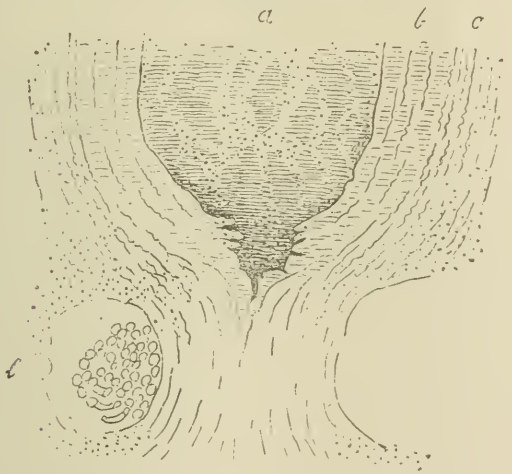
It will be observed that those histologists who have interested themselves in the repair of arteries have considered the question usually from some special standpoint such as the " organization of the thrombus," the role of the white corpuscles, the wandering cells of the endothelium. We will now undertake to follow the various pathological changes which occur in and around the vessel from the time the ligature has been applied until the process of cicatrization has fully completed itself. These changes may be compared not inaptly to those which occur in long bones after fracture. In both we find an external and an internal callus, in both there is a growth of tissue which has only a provisional existence, and in both the dense walls subsequently undergo certain changes which enable them to take a tardy but important part in the final cicatrization. In arteries, however, the external callus produces a ligamentous union only between the two fragments.[1]

When a large artery is tied in its continuity the intima and a variable portion of the media are usually ruptured, and the adventitia is gathered into a dense tendinous sheath around the constricted ends. The first noticeable change is the formation of the thrombi which usually are developed within the first twenty-four hours and the accumulation of a granulation-like mass of cells about the ligature, which if it has been cut short is

[1] Proceedings of the Boston Society for Medical Sciences, 1883. J. Collins Warren.

completely enveloped in them. This growth appears to proceed from the periadventitial tissue, and varies according to the amount of injury done by the ligature to the vessel walls or to the irritation which it produces. If the coats of the vessel have been unnecessarily bruised, and a certain amount of extravasation has taken place in consequence, or the ligature itself for some cause has created irritation, the surrounding inflammatory tissue will form a well-marked callus. If an excessive irritation has been produced the growth of this protective tissue may be retarded, or it may be destroyed and the danger of hemorrhage correspondingly increased. Following the development of this external growth, we find it extends some distance up and down the sides of the vessel in the periadventitial tissue. The round cells of which it is composed invading only the superficial layers of the adventitia, its breadth is of course greatest at the point of ligature; in length it reaches usually to

Fig. 1.



a. Thrombus. *b*. Media. *c*. Adventitia. *l*. Ligature

a point level with the end of the thrombi; when fully developed it is consequently spindle-shaped (Fig. 2). At the point of ligature where the fibres of the outer wall are densly packed (Fig. 1), the wandering cells do not penetrate during the first

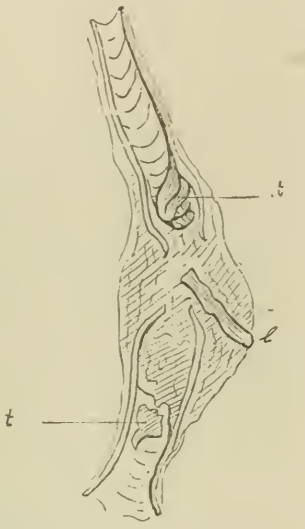
few days, but just above and below the ligature they may be found already invading the media as early as the second day; occasionally the apex of a pyramidal-shaped mass of such cells will have reached the thrombus. These cells appear to exert a solvent action on the bunch of fibres projecting from the ring of the ligature, which thus becomes gradually liberated from all connection with the vessel, the ends of which now retract and leave the knot imbedded in the centre of the callus. The fibres of the ligature soon become infiltrated with cells, and by the tenth day may have already disappeared, or if its resisting powers are greater may remain encysted for some time. The period which the ligature requires for this separation varies greatly according to the size of the vessel and character of the ligature, and longer in man than in animals. If the artery has been properly dissected out this external growth will be observed forming a callus-like ring, in which the two ends of the vessel are imbedded, in size about twice the thickness of the vessel, and can still be seen well developed at the end of two months (Fig. 2).

The question of the organization of the ligature was first raised by Lister, who now explains that he does not mean that the catgut comes to life again, but "new tissue forms at the expense of the old, that the old tissue is absorbed by the new, and that as the old is absorbed, new is put down in its place." Evidently he is here describing the formation of the provisional tissue or external callus, and the gradual absorption of the ligature. In the same sense may the observations of Dent[1] be interpreted; the granulation tissue pushing its way into the interstices of the knot preparatory to "eating it away," as it already has done portions of the muscular coat. Much misconception has arisen from an improper preparation of such specimens. In order properly to appreciate existing conditions the vessel should be removed with the surrounding inflammatory tissue attached to it, should then be placed in some harden-

[1] Medico-Chirurgical Transactions, vol. lxiv. 1881.

ing fluid for some days, and finally a longitudinal section with a sharp razor should divide it into halves (Fig. 2).

Fig. 2.



Artery, two months after ligature, showing external callus —*l*, sinus at site of ligature; *t*, remains of thrombus.

In the specimen from which the accompanying drawing was taken the ligature had caused suppuration about it, and had formed a fistula, at the fundus of which some fibres were found still remaining. By the end of three months the external callus has disappeared, and only a slender cord unites the peripheral to the proximal end.

In rare instances this process is prolonged, as in arterial trunks without many branches like the common carotid. Here the process will not be complete until the granulation tissue within the vessel, as about to be described, has grown up to the first large branch; and in long carotid arteries, like that of the horse, it may be many months longer before the cicatrization has fully completed itself.

Already by the fourth day changes are noticed within the vessel. Observing the proximal thrombus we find an increase in the number of white corpuscles, particularly near the point

of ligature, not in an isolated mass, but mingled with red corpuscles. Masses of fibrine with young cells, white corpuscles of clot, wandering cells from arterial coats, and rarely, also, periarterial tissues are attached to the frayed ends of the media which have been cut by the ligature, and are more or less inverted.

The endothelial cells, when not firmly compressed by the thrombus, as in the distal end, are in a state of activity undergoing proliferation to a moderate extent. Occasionally loop-like masses of cells may be seen projecting into the clot, or a delicate anastomosing network of stellate or spindle-shaped cells, but the total amount of this cell growth is small as compared with the size of the thrombus. Too much importance has been attached to the early changes as indicating the parts mainly concerned in the process of repair.

In the mean time, in the second week, masses of granulation cells are seen infiltrating that part of the wall which is separating or has already separated from the ligature. Even at this period, with the external growth carefully dissected, as is the custom in museum specimens, the vessel appears to have united by first intention, that is, by a direct union of the media and intima side to side. But the infiltration and softening continue until the walls are separated and expand, like the petals of a rose, yielding before the advancing growth of granulation tissue. The deeper portions of the clot are now infiltrated with two growths, the more superficial (that is, the portion nearest the open lumen of the vessel) is composed of tissue derived from the intima and media and wandering cells, and the deeper of vascular granulation tissue, which has pushed its way in from without.

At the end of the third week the extremities of the vessel will be found expanded, and the space between them filled with well-formed granulations, such as are seen on the surface of a healthy wound. A portion of the thrombus, sometimes a large portion, has not been infiltrated, but is attached firmly to the top. A longitudinal section of such a specimen gives a striking illustration of what is understood as "healing by scabbing." As

the clot shrinks the spaces left between the granulations, which have been rolled over one another in cloud-like masses, become continuous with the open lumen of the vessel, and the so-called "canalization" of the thrombus is thus effected. An injection mass can be forced into them from the vessel for some distance, but as yet they do not communicate with the vessels of the granulation tissue.

This does not occur usually until the second month, that is, until the provisional growth has reached its period of highest development. The vessel walls have, in the mean time, been undergoing certain changes. A proliferation of the cells of the intima, as has been noticed by so many observers, unquestionably takes place; the amount developed, however, is not sufficient to supply but a small fractional part of the provisional tissue. The cells, however, have begun to grow before the other tissue has made its way into the vessel, and at this period serve the purpose of attaching the thrombus to the walls of the vessel, but even in this work are aided by cells from the media. They also furnish a new endothelial covering to the permanent cicatrix, and a lining to the new vascular spaces that have been formed. When the elastic lamina has been ruptured, and this is frequently seen on the sides of the vessel nearest the ligature, and also here and there higher up, as far as the thrombus extends, we find an intimate connection at such points of the media with new growing tissue within the vessel. In the second week cells may be seen springing from the media, and growing either into the clot or forming a clump of cells attached to the inner wall. The cells are round and spindle-shaped, frequently in bundles. Evidences of cell activity in the media are abundant, and in some specimens in animals, a proliferation of the muscular cells through the whole thickness of the media is observed, giving a considerable increase to the width of this layer. The elastic lamina is frayed out at its divided end, and glistening elastic fibres are seen extending downwards into the external growth, as the two ends of the vessels retract from one another. At the end of three months the provisional tissue has been absorbed, and we find the walls united by a permanent

cicatrix which join the sides of the vessel still somewhat separated from one another. It consists in medium-sized arteries of a crescent-shaped mass of tissue, the concavity presenting to the lumen; the horns running up on either side of the vessel. One horn may be long and the other short, the crescent being placed somewhat eccentrically. The larger horn may be sometimes thickened, as in Fig. 3, if a branch lies opposite to it. In the largest vessels the cicatricial tissue occupies a considerable portion of the calibre of the vessel.

On the surface of the cicatrix is seen a thin layer of endothelium; beneath this, in medium-sized vessels, there can be seen

Fig. 3.

Fig. 4.

Fig. 3. Human carotid artery four years after ligature, showing nearly complete obliteration from aorta to bifurcation.

Fig. 4. Femoral artery of a dog three months after ligature.—*a*, new muscular layer; *b*, media; *c*, adventitia; *d*, cicatricial connective tissue.

a layer of delicate, tapering spindle-cells with staff-shaped nuclei forming a continuous layer from one horn to the other. They are parallel to one another and to the arc of the circle made

by the crescent, they resemble in all respects muscular cells, in short a genuine muscular layer is found here. Beneath this layer is a mass of cicatricial connective tissue, which plugs the space lying directly between the ends of the retracted walls. The cicatrix is pierced by a vessel of considerable size, which rapidly tapers to a point, and anastomoses with a capillary network, ramifying both in the cicatrix and in the ligamenture band outside. This central vessel, which in larger cicatrices becomes tortuous and gives to the cicatricial tissue a "cavernous" appearance, may be regarded as the unobliterated residuum of the lumen. In such a specimen as Fig. 3 represents, this crescent-shaped muscle is not apparent as a separate layer, but its equivalent may be traced with the microscope, as bearing a similar relation to the vascular spaces surrounded by the cicatrix. The minute vessels of the cicatrix resemble in all respects those usually found in cicatricial tissue.

The novelty of the theory of the participation of the muscular cord in the formation of the permanent cicatrix has led me to study the action of the muscular cell with special care. Cells of this character may be found in the cicatricial tissue of arteries of horses, and in man both after ligature in continuity and in amputation stumps. The proliferation of muscular cells may also be seen in the cicatrix formed by the closure of the ductus arteriosus when examined at different periods of its development. In the disease known as obliterating endarteritis these cells are quite numerous, being found both as longitudinal cells in the newly-formed tissue, and forming circular bands around the new vessels seen in this tissue, some of which are of considerable size.

In the main artery of an amputation stump we do not find the lumen terminating in a cul-de-sac as in Fig. 4, but a condition analogous to what has been called a compensatory, and in some cases even an obliterating endarteritis. That is, the calibre of the vessel is not abruptly terminated, but gradually loses itself in a fine network of branches. When the trunk is a large one, as the femoral, it will still remain as a solid cord in which new arterioles are to be found ramifying.

We find in this anatomical peculiarity of the cicatrix an explanation of its immunity from aneurismal dilatation. The protective influence of the thrombus enables the process of cicatrization to complete itself before it is called upon to withstand blood-pressure, and it is then armed with a muscular coat, like the normal vessel wall, acting not unlike a levator ani muscle in sustaining and modulating the force of the blood column.

The ligament which unites the two ends of the vessel after ligature in continuity, represents in part the residue of the external callus; it has become much elongated by the retraction of the two ends. During the healing process a small portion of the vessel walls has become disintegrated, and a portion has atrophied, and has been absorbed; the remaining walls have shrunk greatly by retraction, and their calibre has been filled to a greater or a less extent by a cicatricial tissue, so that the vessel has been practically obliterated up to the first branches of the collateral circulation.

When a double ligature is placed upon a vessel and the intervening portion allowed to remain intact, it is, by the processes above described, gradually absorbed granulation tissue, growing in at both ends as the fragment is separated from the ligature, and finally infiltrating the whole mass. It may be possible at the beginning of this process to demonstrate the passage of a few wandering cells through some injured spot, during the first forty-eight hours, or to verify the proliferation of certain endothelial cells within the segment; but they, like the other elements of this isolated mass, have no further function to perform and are eventually destroyed.

The thrombus is a mere passive structure, takes no part in the growth, but is protective, and affords an excellent medium for the new tissue to germinate in. Its upper portion is not penetrated by the new growth, but rests upon it and forms a sort of protecting scab. It is deposited gradually, and has in large vessels a stratified appearance. The size appears to be dependent upon the amount of injury done to the vessel and the subsequent inflammatory process. If the intima or the elastic lamina have been ruptured in handling the vessel, as can frequently be

demonstrated by the microscope, some distance from the point of ligature, clot will form at those points and the thrombus become thus elongated. Occasionally the apex is formed of white corpuscles only, and may be free or lightly attached to the wall by a cell-growth from the intima. The distal thrombus is shorter than the proximal and much narrower, so that there is but little difficulty in distinguishing them from one another. In illustration of the views advanced, the following specimens are submitted to the inspection of members of the association.

The first series of three bottles contains specimens from the common carotid of the horse. After removal together with the enveloping callus from the subject they were placed in alcohol, a weight being attached to the end of each to keep them straight. Subsequently they were divided into longitudinal halves by the razor. At two weeks we see the ligature embedded in a large spindle-shaped callus which surrounds the ends of the artery. The ends have not yet perceptibly retracted from one another. The calibre of the vessel is filled with a stratified thrombus.

At two months we have the specimen for which Fig. 2 was taken.

At four months we have still some of the external callus, but the clot has been replaced by a new growth of tissue, growing in at the ends apparently. The first large branches are some distance from the ligature, and it is probable that some months will elapse before the intervening artery will become obliterated. In specimen No. 3983, Army Medical Museum, you see the subclavian tied for gunshot injury and subsequent aneurism of the axillary artery. The patient died on the 57th day. The process of repair is much more advanced at the distal end which is close to a larger branch than at the proximal, which is at about the same stage of repair as shown in Fig. 2. The callus unfortunately has been dissected away.

Specimen 3986 shows the external iliac 129 days after ligature reduced to a cord. The removal of the wall at the point of insertion of this cord at the junction of the common and internal iliacs shows a beautiful cicatricial tissue occupying the shallow cul-de-sac which represents the origin of the external iliac.

Complete cicatrization has taken place and the entire external iliac has been reduced to a cord within four month's time. In the microscopical specimens also presented, you will be able to verify the various points dwelt upon in this paper.

DISCUSSION.

Dr. N. Senn, of Milwaukee, Wisconsin.

I am sure that we all feel grateful indeed to Dr. Warren for these beautiful illustrations and this demonstration of specimens, and more particularly for the new view advanced in regard to the part which the muscular tissue takes in the process of cicatrization. I have little to say, inasmuch as my views on the subject of cicatrization in bloodvessels after ligature were incorporated in my paper read at the last meeting, but I shall make a few remarks respecting the behavior of the tunics of a vessel after ligature. From the illustrations and specimens presented, it is evident that a maximum amount of traumatism was inflicted in applying the ligature. It is apparent also that aseptic precautions were not exercised, inasmuch as all the specimens and illustrations plainly indicate the process of regeneration in excess of that which is necessary to obtain the ideal result after ligature. In the vessels, I believe, that we are all satisfied that the substance which yields the granulation tissue is produced by the connective tissue and its derivatives. These tissues during the process of cicatrization are reduced to the embryonic state, producing granulation tissue which infiltrates the tunics of the vessel walls and the thrombus, if this has been formed. All that is necessary to obtain definitive closure of a vessel is simply to approximate the intima without rupturing it, and keep it in uninterrupted contact until cicatrization has taken place.

In the first illustration, it is not only the intima which has been ruptured, but also the media. This would naturally explain why so many colonies of migration corpuscles were found in the interior. Such an amount of traumatism is not only unnecessary, but in fact harmful. The ideal result after ligature is simply to bring the parts into approximation and keep them there until the granulation pro-

cess is completed. This requires a comparatively short time. The behavior of the tunics depends, in the first place, on the nature and quality of the material used for ligature; in the second place, on the extent and intensity of the inflammation induced by the ligature. After ligature of a vessel in any portion of the body, an unusual response on the part of the tissues takes place when suppuration is established, so that the formation of a maximum amount of callus or cicatricial material is no guarantee against secondary hemorrhage. In fact, in my experiments, I found that where I failed with antiseptic precautions, there was an enormous amount of external callus, and although the endothelial proliferation did not keep pace with the regeneration of the perivascular tissues, yet secondary hemorrhage was never observed. We are all aware, however, that in the human subject secondary hemorrhage may take place at any time, if suppuration follows the operation no matter how abundant the external callus may be.

If you ligate a vessel with a temporary ligature, capable of being removed by absorption as soon as its temporary purpose is fulfilled, as, for instance, aseptic catgut, you secure permanent obliteration of the lumen of the vessel without separation of any of its tunics. The ligature holds the parts in contact, while the granulation process goes on. In from seven to twenty days its place is substituted by granulation tissue and it disappears by substitution without producing a loss of continuity of the vessel.

This is, however, different, if the vessel is ligated with a permanent ligature, which is incapable of being absorbed. Under such circumstances, even if the tunics are not divided, the pressure produced by this inelastic substance gradually interferes with the vitality of the parts beneath, the tissues are absorbed by the granulation tissue, or in the event of suppuration, necrose, and are expelled with the products of suppuration.

By an ideal ligature we mean a ligature which is capable of being absorbed, and which keeps the parts in uninterrupted apposition until obliteration has taken place.

Dr. Warren has compared the process of cicatrization of a vessel to the regeneration of a bone after fracture, a very happy comparison. I shall make a still further comparison and say that the ideal result in the treatment of fracture is to treat the bone in such a manner, that only a minimum amount of callus is necessary to restore the continuity of the bone. We are all aware that if the fragments

are kept in uninterrupted contact, we have a minimum amount of callus. If the fragments cannot be placed in exact apposition, or cannot be retained in uninterrupted apposition, an abundant callus production takes place. Nature comes to the assistance of the surgeon, and renders material aid in preventing unfavorable results. In the application of a ligature we should aim to obtain only a minimum amount of callus, or cicatricial material, and this will invariably take place, if you apply a temporary ligature under strict antiseptic precautions. A minimum amount of material is all that is required in the definitive closure of a vessel. I believe my experiments with the double ligature prove that where we have no thrombus, no blood clot, which would answer as a culture material, that direct, immediate union takes place in a remarkably short time between the layers of the intima by infiltration of granulation tissue through the walls of the vessel and from the intima, so that only a minimum amount of cicatricial tissue was necessary to effect obliteration. The early definitive closure, without regard to external callus is what we want to obtain. The latter is desirable where we fail in obtaining primary union. If it is necessary for the ligature to cut its way through, or, in the event of suppuration, the external callus may be of much assistance and may prevent secondary hemorrhage.

I now come to the most interesting part of Dr. Warren's paper, and that is the presence of muscular tissue in the cicatrix. This idea is certainly a new one, and I hope that future research will show the correctness of this observation. All that I can say, this observation lacks analogy. When a muscular fibre is divided anywhere in the body, we know that the union which takes place is established through the medium of the connective tissue between the muscular fibres. As far as I am aware, there is no such thing as direct union after section of a muscular fibre. In fact, all wounds, wherever inflicted, are repaired through the medium of connective-tissue proliferation.

Dr. Warren has said that he has wondered why we have not to deal more frequently with aneurism after ligature. I would say in the first place, that after ligature of an artery, the tissues in proximity to the ligature are deprived of their physiological function. Hence rest is established, either by the formation of a thrombus in its lumen, or subsequently by the definitive closure of the vessel. What is the result of placing a muscular fibre at complete rest? I have invariably observed that the muscular fibre undergoes fatty

degeneration, and is removed by absorption, and its place is occupied by connective tissue. Why should this not be the case in vessels? What prevents the formation of aneurism, is the development of connective tissue, unyielding in its character, admirably adapted in every respect in resisting intra-arterial pressure vastly more effectively than would be the case if the cicatrix contained muscular fibres. I would like to ask Dr. Warren if it is not perhaps possible that the muscular tissue found in the terminal end of the cicatrix might not be muscular fibres, which pre-existed and had been dislocated by cicatricial contraction? I hope that he will be able to prove his point, but certainly it seems to me that nature would only err, if the observation were proved to be correct. I believe that it is the unyielding cicatrix and the absolute rest which is obtained by a limited portion of the vessel that secures the patient against the occurrence of aneurism.

Dr. Moses Gunn, of Chicago, Illinois.

This paper, like the one presented last year on the same subject, is of intense interest to the practical surgeon as well as to the histologist, perhaps possessing even more interest, inasmuch as practical results are more directly interesting than the routes by which we travel to reach these results. These studies are very interesting to us as applied to the ligature of vessels of large size. As a matter of course, the great danger that has always been encountered in the ligation of large vessels, is secondary hemorrhage. The old method was that the ligature was to be applied tightly, or as Liston used to say: "How tightly? He didn't know, but devilish tight." Force enough was used to lacerate the intima and the media and bring the adventitia in contact. This necessarily diminished the strength of the vessel and made a weak point, and at that weak point, it was no wonder that in certain instances, where we had recurrent forces established, the vessel would give way. The natural question was, How can we secure coaptation, adhesion, and cicatrization of the vessel with the least amount of destruction of its coats? This has haunted my mind for the last twenty-five years. I have been waiting in vain to get at an innominate artery. As long ago as I have mentioned, I devised in my mind a plan like this. A ligature around the innominate artery, applied in the ordinary way, so tightly as to lacerate the internal and middle coats, would necessarily make a weak point there, and when the recurrent circu-

lation was established and the weight of the current came down through the vertebral and external carotid and its force was brought to bear upon this point, it would give way. I said to myself (this was before the days of antiseptic surgery): If we can get this vessel obliterated and early union of all the soft parts, we may avoid the danger of secondary hemorrhage. I made up my mind, if good fortune ever sent me an innominate artery requiring ligature, I should cut down upon it and reflect the tissues with as little dissection as possible, turning them back in one mass if possible. I would then throw around the vessel a silver wire of large size, as large as that used in wiring bones together. This I would twist sufficiently tight, but not enough to lacerate the vessel, and without constricting the vasæ vasorum, without doing anything to injure nutrition. After twisting it sufficiently to secure coaptation, I intended to compress the wire so as to make the two sides parallel, having the loop on the deep side and the twist on the superficial. This would afford a permanent mechanical obstruction to the vessel. I would then close the wound and get union by first intention, if possible. I held this idea in regard to this particular vessel until one year ago, when I listened to the paper of Dr. Senn, who claimed that we should secure coaptation with as little injury as possible to the vessel, and that his experiments pointed to the fact that it was not necessary to lacerate any of the tunics of the vessel, but to bring them into coaptation and then by a process of proliferation, the walls would become consolidated, and when he advanced the idea of his double ligature, in which after applying one ligature, then emptied the vessel of its contents and then applying the second ligature so that the walls of the vessel were lying in contact, I thought that it would be an admirable plan for ligating the innominate artery. Throw a ligature, catgut if you like, around the artery and draw it sufficiently tight to obstruct the calibre of the vessel, without lacerating any tissues. Then throw a ligature around the subclavian at its commencement, and another around the carotid, but before tying them, empty the vessel of blood, thus leaving the two sides in coaptation. In this way we may reach a point where our hopes may be realized. Then came the thought of the recurrent force down the vertebral artery, acting on the last vessel in the same way, but still if the ligature can be applied without lacerating the tunics, the vessel would be in a condition to resist that pressure. The walls of the vessel, undiminished in strength,

would be in a condition to resist that pressure, until the reproductive process had become complete.

Dr. Warren comes to-day showing us a series of illustrations and actual specimens where the ligature is applied in a more destructive manner, and shows us a process more complex, by which union has been established, but in addition to anything that has been said before, he has shown us an additional barrier which is thrown up by what he supposes to be a new production of muscular tissue. That is a point which devolves more particularly on histologists. To me and to all practical surgeons, the whole interest depends upon this one thing, securing union as rapidly as possible and securing it with as little injury to the vessel walls as possible, so that we may have the strength of the vascular walls to aid us and nature in preventing secondary hemorrhage while consolidation is taking place.

I must confess that I think that I see more clearly from my standpoint, to the desired result in such an application of ligatures as will fulfil the indications of which I have spoken, that will obsstruct the vessel without inflicting any injury to the vascular tunics.

Dr. WARREN. I shall not reply at any length to the observations which have been made, but there are one or two points which I should like to answer. In the first place, I agree entirely with the view expressed by Dr. Senn, that the amount of this inflammatory provisional tissue depends on the amount of traumatism. I have selected some of the most marked examples, simply to emphasize the point which I raised. Frequently it is difficult to see the external callus. Even under the most strict antiseptic precautions, callus of this sort is seen. If you will remember, Lister, in studying the organization as he thought of his catgut ligature, said, that when the artery was dissected out after being tied, he found a ring of new tissue around the vessel. This ring of new tissue, which he supposed to be the catgut converted into living tissue, was undoubtedly nothing more than this ring of external callus.

One of the chief points of Dr. Senn, and it has been referred to by Dr. Gunn, is the question of primary union. When I first began to make experiments on the ligature of vessels, I was inclined to adopt this view. I had specimens showing the walls in approximation at the end of the tenth day. After going back to this series of specimens, after studying the complexity of the process, I found cases where there was a minimum amount of thrombus, almost

none. The thrombus was very small, being only a little clot of blood attached to the vessel. It seemed to me that here was an instance of what Dr. Senn called primary union. If we look at the edge, we find some endothelial cells heaping themselves up. I supposed that that was a case of primary adhesion. A closer observation showed that in the walls of the vessel the process of disintegration was going on from the gradual invasion of granulation tissue from without, and this was only the end of the first stage of healing.

As regards the question of the closure of a vessel without laceration of the intima. The pathologists who have studied this subject and the coagulation of blood, as for instance Cohnheim, have shown that, while blood flows through a perfectly uninterrupted vessel, there is no tendency to coagulation, even if it flows slowly or stands still.

If the intima is not ruptured, the blood remains fluid. The moment there is any abrasion, a thrombus forms. The idea of thrombus from stagnation is being accepted less and less, while the idea that it comes from an injury is more and more accepted. Just in proportion to the bruising do we have internally the protective thrombus, and externally the protecting callus.

I have one specimen of ligature of the common carotid, in which, at the end of fourteen days, no tendency toward union seems to have taken place. The intima was shown to be continuous. There had been separation of the external coats of the vessel, but the invading cells had been unable to get through the uninjured intima, and there was little thrombus in the interior. The cells had dissected the intima from the outer coats. There seemed to be a process of disorganization, but an unhealthy process, which would tend toward secondary hemorrhage. The intima was doing the holding. This was in the case of a poor girl who died with cancer. It struck me that the observation was a suggestive one.

In regard to the absorption of ligatures, even large ones are absorbed. I have used cotton thread in all cases, and in two or three I have found that the ligature was disintegrated and destroyed. After all, most thread is of an animal character.

In regard to the point of Dr. Senn in regard to the fatty degeneration of muscular fibre and the non-formation of a muscular cicatrix. We must make an important distinction between striated and non-striated muscular fibre. The two are very different. The non-striated muscular fibre is a spindle-shaped cell. The striated is

much more complex, and this never unites. The cicatrix of striated muscle is connective tissue. What happens in a granulation wound? New arteries are thrown out provided with new muscular walls. In the uterus we have marked changes in the muscular fibre, as seen in pregnancy and in myoma. There we have a good illustration of the production of new unstriped muscular fibre. A considerable portion of the walls of the vessel undergoes absorption, as I have shown in the carotid artery of four years' standing, where the vessel is reduced to a cord.

In regard to the approximation of arteries, something which Dr. Gunn has suggested has interested me. It seems as though we could arrive at some plan of applying the ligature by which we might gain some advantage. The more I have thought of it, the less have I been able to come to any conclusion. In Ashhurst's *Surgery* the ox-aorta ligature is spoken of. This is cut into strips and stretched by a weight. The idea is to approximate the wall of the vessel without breaking it, giving much the condition which we obtain when we apply a tourniquet to shut off the artery until coagulation of blood may take place in an aneurism. Where a ligature is permanently applied, I am confident that we shall have changes of the character which I have indicated. If antiseptic precautions are preserved, they may be so slight as to almost escape observation; but if there is a great deal of suppuration, or danger of the ligature slipping, a large barrier is thrown out, and surrounds the ends of the injured vessel and protects it until suppuration has subsided. I think that under any conditions which have been devised, the process which I have sketched is more or less perfectly carried out.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

I should like to ask Dr. Warren a question. Dr. Senn in his paper last year regards the clot as having no purpose in the closure of the vessel, in fact looked upon it as rather in the way. Dr. Warren reduces it to a minimum at least. I should like him to express himself in regard to the value of the clot in the process of closing the vessel against this danger of possible secondary hemorrhage. In a case of ligation of the lingual artery, secondary hemorrhage occurred, and, in my absence, the man bled to death. In examining the vessel, I found an opening just behind the ligature, without the slightest indication of any clot in the vessel. I

should like Dr. Warren to give more light upon the value of the clot in the process of closing the vessel and in preventing secondary hemorrhage.

Dr. Warren. My point was that it took no active part in the process. I think that we cannot ignore the value of the clot. Anything that favors the development of a thrombus to a certain point is of advantage. In small vessels and in the distal extremities of some, I presume that it is not of great value. Where the blood current is strong, it is important that rest should be given by the protection of this thrombus. It is a provisional structure. It is the first stage of the internal protective callus, and protects the vessel during the early stage.

Dr. Edward M. Moore, Rochester, New York.

(To Dr. Warren.) Did I understand you to state that Lister said that the catgut ligature was converted into tissue?

Dr. Warren. Yes, sir.

Dr. Moore. I am defending Mr. Lister. It was impossible for him to say anything so outrageous. I know where you got your information. Dr. Hodgkin wrote a paper in 1876, when Mr. Lister was here. He had quoted this idea, and it was down in a book. The reporter had put it in a book. When it was announced before Mr. Lister, he called out (English fashion), "Hear," and renounced the idea that he could say anything so absurd as that catgut could be converted into living tissue.

Dr. Warren. Not only has Mr. Lister made the statement in print, but others have taken up the idea and carried it out. Mr. C. T. Dent has printed a lithographic plate showing the organization of the ligature. The specimens exhibited to-day give the correct interpretation of the process which is the formation of an external callus.

SOME POINTS IN THE SURGERY OF THE HYPERTROPHIED PROSTATE.

By JOHN W. S. GOULEY, M.D.,
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Since the time of Sir Everard Home, the advances that have been made in our knowledge of the anatomy, pathology, and surgery of the prostate, are due mainly to the indefatigable industry and the genius of the late Dr. Mercier, of Paris, who began his labors in 1836, and afterwards published their results in a series of volumes with the modest title of researches, etc., on diseases of the urinary organs of elderly men. These works bear the stamp of consummate ability and literary acumen, and honest, diligent research. They are vast treasures of anatomical and surgical knowledge of the subjects which they treat, and require to be carefully and patiently studied, not casually read. Those interested in the study of the infirmities of old age will almost always find in them something suggestive or likely to shed light on a doubtful point.

Until about five years ago, Dr. Mercier was actively engaged in study and in professional work, but bodily infirmities together with advanced years have put an end to his labors which, fortunately for the profession and for the aged, are so faithfully and honestly recorded in the treatises which he has bequeathed to the profession. There is little to be added to the principles he has inculcated; and many procedures which may seem new are in reality only modifications of his devices.

The importance of thoroughly studying the pathology and therapy of the hypertrophied prostate will be fully realized if the conclusion of Mr. Messer, of London, is accepted; *i. e.*, that thirty per centum of men upwards of fifty-five years of age are

affected with enlargement of the prostate, although he also says that only one in seven of those so affected suffers in consequence. Mr. Messer has probably underestimated the number who really suffer some inconvenience in urination. It is, however, a fact of no little interest and practical importance that even when enlargement of the prostate causes stagnation of urine in the bladder, the patient's life is not, on that account, materially shortened if the catheter be properly, persistently, and symmetrically used as soon as indicated. But the omission of this precaution is invariably followed by the most disastrous consequences.

The particular points in the management of cases of senile hypertrophy of the prostate and its consequences which I have the honor to suggest for discussion relate to—

- A.—Physical exploration.....
  - *a.* Digital examination per rectum.
  - *b.* Urethro-vesical exploration with catheters and sounds.
- B.—Medical treatment.
- C.—Surgical treatment....
  - 1. By mechanical means
    - *c.* Evacuative catheterism.
    - *d.* Irrigations to relieve cystitis.
  - 2. By operation.....
    - *e.* Prostatotomy, internal and external.
    - *f.* Prostatectomy, internal and external.

A.—*Physical Exploration.*

a. By digital examination per rectum some estimate of the size and consistency of the prostate can be formed, and it is possible to ascertain if this body is harder or softer than normal, if one lobe is larger than the other, if it is nodulated or smooth and uniformly enlarged, if its apex is obtuse instead of being insensibly lost in the membranous portion of the urethra, or if there is longitudinal increase. This is all that can be learned by digital examination, which is only the first step toward complete physical exploration. Many decide at once upon a case without carrying the examination farther, and are thereby misled and deceived, for it often happens that no enlargement is detected by rectal examination when there exists sufficient urethro-

vesical obstruction to cause stagnation and finally complete retention of urine. Therefore the surgeon should never place his entire reliance upon rectal examination.

b. The next step in the physical examination of a case of suspected prostatic hypertrophy is to ask the patient to stand up and urinate; if he can urinate spontaneously, the character of the jet of urine will aid to a certain extent in the diagnosis, but it is to be borne in mind that a man who has no prostatic hypertrophy, but contracture of the vesico-urethral sphincter, urinates precisely in the same way as he with enlarged prostate, *i. e.*, in a small, weak, and perpendicular stream, suddenly interrupted and soon followed by a succession of drops, perhaps twenty or thirty, to begin again, and to be again followed by the dribbling or even slobbering until two or three ounces of urine may be collected in a vessel, without which the patient surely soils his garments or wets his shoes. But the digital exploration per rectum may have cleared the doubt, for general hypertrophy may have been detected.

When the patient has passed all the urine he can void spontaneously, a soft catheter is introduced, and the residual urine drawn off, measured, and inspected. It may be clear or turbid. The turbidity may be owing to the presence of gravelly substance, of pus, or of both. Such purulent urine indicates cystitis; ammoniacal, slimy urine, decomposition of the pus, which proves that the urine has been stagnant for a considerable time. This condition is improperly called catarrh of the bladder. Fetid, slimy, chocolate-colored urine indicates that hemorrhage has occurred, and that the cystitis is in an advanced stage.

Residual or stagnant urine in the bladder is then another link in the chain of evidence of prostatic obstruction.

The physical exploration is, however, further carried on by the use of certain rectangular metallic catheters and sounds (instruments of ordinary curve, possessing no value whatever for purposes of diagnosis and being even dangerous), with a view of determining the situation, size, or form of the obstruction. To Dr. Mercier belongs the credit of suggesting and first employing the short-beaked, rectangular catheters and sounds for ex-

ploring the prostate and bladder, as well as for evacuative catheterism in retention of urine. He may have taken his cue from Benjamin Bell, who had used a slightly angular bougie in cases of eccentric urethral strictures. The advantages of the rectangular beak over all others for the uses to which it is applied are: 1. The extremity of the short beak (which is seven-eighths of an inch in length) is constantly in contact with the upper urethral wall in the prostatic portion. 2. The blunted heel comes in contact with the obstruction, and overrides it easily, while in the case of the long curved instrument the point is presented to the obstacle. 3. The beak can be rotated with the greatest facility when it has entered the bladder, and serves at the same time as a stone searcher and as the means of detecting induration and hypertrophy of the bladder. Thus, by the aid of the rectangular sound, it is possible to diagnosticate intra-urethral hypertrophy of either lobe of the prostate—the deflection of the handle of the instrument indicating the affected side—longitudinal hypertrophy, intra-vesical hypertrophy of one or both lobes, supramontanal hypertrophy, whether as a crescentic valvule, as a bar, or as a sessile or a pedunculated tumor or tumors. Dr. Mercier has sometimes been able to determine the size of such tumors.

A few years ago it occurred to me that an instrument of still greater precision might be constructed. I accordingly devised a cysto-pylometer, by the aid of which I have been able to measure accurately the thickness of crescentic valvules and of urethro-vesical "bars."

The beak of this instrument is nearly rectangular, and one inch in length. The shaft is in two longitudinal segments, the male segment sliding over the female, and being flat at its vesical end to come in contact with a correspondingly flat surface on the inner side of the beak, and designed to grasp the valvule or the bar. The reverse of the flattened, hollow, and quadrilateral handle, is graded in centimetres and millimetres, and the indicator is screwed to the shaft of the male blade. The pylometer can be used at the same time as a stone searcher.

B.—*Medical Treatment.*

The idea that certain medicinal agents, taken by mouth or otherwise, can reduce the enlarged prostate, though long since exploded, is from time to time revived on account of the supposed discovery of some infallible remedy. To "cure" prostatic hypertrophy, all the most likely drugs in and out of the Pharmacopœia have been exhausted in vain. Thousands of sufferers have been deluged with innumerable natural and artificial mineral waters with the very worst results. Others who have taken the waters in small or moderate quantities have, though indirectly, suffered seriously on account of having omitted the essential and rational treatment of their complaint, and of using the waters exclusively under the erroneous impression that they possess curative powers.

A few years ago it was the fashion to inject into the substance of the prostate solutions of ergotin, iodine, and of other irritants. Abscesses and their consequences were often the only rewards of these experiments.

Electricity, applied through the urethra or rectum, has had its day, and has also been found wanting, and often positively harmful.

This catalogue of therapeutic agents might be lengthened almost indefinitely, and give the same history concerning results as that which precedes and which leads to the conclusion that medicinal treatment is of no use whatever, and is delusive, and therefore injurious when employed with a view to reduce hypertrophy of the prostate. But when it is applied to relieve some of the consequences of prostatic obstruction to urination, to combat concomitant affections, and to improve general health, it is, in all three particulars, of great value in conjunction with the surgical treatment.

The principal concomitant affections, complications, and sequelæ which the surgeon is called upon to treat medicinally, are dyspepsia, lithuria, costiveness, constipation, sciatica, and other neuroses, urethral fever, cystitis, pyelitis, pyelonephritis,

polyuria, oliguria, anuria. The details of medicinal treatment need not here be given.

C.—*Surgical Treatment.*

The surgical treatment of prostatic hypertrophy and its consequences may, with propriety, be divided into two distinct parts. First, that which relates to the mechanical means of relief of obstructed urination and its first consequence, cystitis; second, that which relates to the removal of the obstruction by operation.

1. *Mechanical Means of Relief.*—Before proceeding to discuss the mechanical means of relief, permit me to say a word about catheters, and first let me urge that all rigid catheters be discarded, except in cases of false routes, where Hey's and Mercier's methods are clearly indicated. The promiscuous and careless use of rigid instruments, whether metallic or non-metallic, is almost certain to lead to the infliction of wounds in the urethra, either at the bulbo-membranous region or in the prostatic sinus, and these rents soon after may be converted into troublesome false routes, which often render further catheterism of the bladder impossible, and place the patient's life in great jeopardy. Even when used with the greatest caution these catheters frequently do much mischief.

Among the flexible instruments now in use may be mentioned the soft vulcanized India-rubber catheters, the olivary and curved gum catheters, and the elbowed and double-elbowed catheters of Mercier. The various deformations of the diseased prostate will suggest the kind of catheters to be used. Thus, for instance, in the case of moderate supra-montanal hypertrophy, the curved gum catheter will answer, but a greater degree of the same hypertrophy will require the elbowed or even double-elbowed catheter. Intra-urethral deformation, causing tortuosity of the urethra will indicate the use of the olivary catheter, or of the soft India-rubber catheter, while great longitudinal hypertrophy or the presence of a large tumor in the supra-montanal region will render necessary the employment of an extra long catheter.

Each catheter is best for the case in which it is indicated, and no particular instrument can be used with equal safety or success in all cases.

With your permission I shall make a few quotations from a paper on chronic retention of urine in elderly men, which I read before the Kings County (N. Y.) Medical Society in 1882.

"I am sometimes asked what is the proper catheter, what kind of catheter should be used by those who cannot urinate owing to a urethro-vesical obstruction. My answer is invariably that no one kind of catheter is THE catheter. The same catheter will not suit all cases any more than the same glove will fit all hands." . . . "Let me express my own convictions as to the catheters the use of which should in general be avoided. All metallic and very rigid non-metallic catheters or soft catheters armed with stylets should not be used under ordinary circumstances. If such instruments had never been employed to relieve retention of urine from prostatic enlargement, cases complicated with false passages, now so common, would be of extreme rarity. I have never owned what is usually called a silver prostatic catheter, nor have I ever seen a case which I could regard as requiring such an instrument. The difficulties which arise from catheterism with metallic or rigid non-metallic instruments are encountered: 1. At or near the bulbo-membranous junction of the urethra from an inordinately deep sinus of the bulb, undue force causing the point of the catheter to perforate the mucous membrane and enter the spongy tissue, or to plough its way between the membranous urethra and rectum, and sometimes even enter the intestine. This accident makes itself manifest by a more or less copious urethrorrhagia. 2. In the sinus of the prostate. This latter impediment is formed by a urethro-vesical barrier, causing a deep pouch in the prostatic sinus. In this case the same undue force may give rise to a longitudinal rent on one side or the other of the floor of the prostatic sinus, extending to or into the barrier, or else the point of the catheter penetrates and sometimes transfixes the barrier at its centre. Such serious injuries are not generally inflicted with soft non-metallic instruments, though when their eyes are

improperly constructed they cause considerable irritation and even erosion of the mucous membrane. Therefore, one of the prime requisites of a good catheter is an eye as small as practicable, with perfectly smooth, rounded edges. It should never have two eyes. The size of the catheter is also worthy of consideration. Nearly all those who practise auto-catheterism have a predilection for small catheters, such as Nos. 4, 5, 6 English. This is a grave error. The catheter should seldom be under No. 9, which, though it may be pliable, retains a certain degree of firmness, and can be much better managed. If it seems too large the surgeon should dilate the urethra. Indeed, it is an excellent practice every few months to put the patient's urethra through a short course of dilatation to No. 13 or 14, if a No. 9 is afterwards to be used, as this canal will contract from inflammation induced by the five or six daily catheterisms when these have been continued for months or years. In this condition of stenosis the introduction of the small catheter will become more and more difficult, if not impossible, and will cause hemorrhage and other mischief. In cases where false routes have been made I have advised the use of catheters as large as No. 15 English, which the patients are now introducing with the greatest ease, while before they had constantly failed with smaller instruments. Of course, in giving such advice the surgeon must be guided by the normal size of each patient's urethra."

When catheterism is to be intrusted to inexperienced persons, to nurses, or to the patients themselves, the soft vulcanized India rubber instruments are unquestionably the safest, but when they fail it is not wise to leave other catheters in their hands until they have been taught how to use them, and are made aware of the dangers attendant upon careless or rough manipulations.

In cases where ordinary catheterism has been impracticable, owing to very large supra-montanal tumors, or to false routes, I have often known Hey's[1] method to succeed; but when this fails, in the case of false routes, the invaginated catheter of Mercier is almost certain to be successful.

[1] Dr. Dorsey claims priority for Dr. Physick over Mr. Hey in this method of catheterism. See Dorsey's "Surgery," page 161.

On five or six occasions, having entered the bladder with the male part of the invaginated catheter, I gradually and cautiously pushed together onward into the bladder both the male and the female portions of the catheter, and thus made free and complete divulsion of the false route, and gained almost as much as by an internal prostatotomy, and the patients have been able to urinate spontaneously, and empty their bladders. The after-treatment was the same as that employed in internal prostatotomy.

Evacuative catheterism cannot be employed too soon in the management of prostatic obstruction. In ordinary cases it is an easy process attended with immediate relief, and can be repeated daily once, twice, or oftener, according to the requirements of the case. But there are cases where it would be most unwise in the surgeon and often fatal to the patient to withdraw all the urine retained in the bladder at one sitting without taking certain steps which I shall presently describe. But before doing so I should like to give, on this subject, the views of a great master. In his *Lectures on the Diseases of the Urinary Organs*, second edition, London, 1835, pp. 159, 160, Sir Benjamin Brodie says: "However necessary it may be in all cases, there are some in which much discretion is required in resorting to the use of the catheter. . . . If, in a case of chronic enlargement of the prostate, the patient has been allowed to go on for two or three years, or longer, without the use of the catheter, and, in consequence of this neglect, the quantity of residuary urine in the bladder has gradually increased, so that at last one or two or more pints are accumulated in it, the kidneys having at the same time become diseased, the introduction of the catheter, according to the rules formerly laid down, so as to empty the bladder two or three times daily, is likely to be injurious rather than beneficial. The patient is, it is true, relieved of many of his distressing symptoms. He is no longer tormented by a frequent desire to void a small quantity of urine, nor by an involuntary dribbling of urine during the night, nor does he suffer the uneasy sensation which, in a greater or less degree, always attends an over-distended bladder; but, in the course of

a few days, it is observed that he avoids his usual exertion, that he seems languid, and loses his disposition to take food. Then the other symptoms of disease of the kidney, which were but imperfectly developed before, become distinctly marked, and he gradually sinks and dies at the end of a month or six weeks from the time of the catheter being first employed." He then draws a parallel between this and "the consequences of the operation of lithotomy performed on a person who labors under any considerable disease of the kidneys. At first he is greatly relieved, and often seems to recover rapidly from the effects of the operation; but, in the course of a few days, his bodily powers begin to fail, and death ensues at no distant period, and this happens even where the stone has been of a small size, extracted in the shortest possible space of time, and with the least possible injury to the parts concerned. The resemblance between the effects produced by the use of the catheter, in the way, and under the circumstances which I have just endeavored to describe, and those which follow the operation of lithotomy in a patient similarly circumstanced, is too obvious to be overlooked, and I conclude that they are to be referred to a common principle. The system suffers from the shock of the operation, in one case, and in the other case it suffers in the same manner from the impression made on it by the sudden emptying of the over-distended bladder, and consequent removal of the pressure which is made through the medium of the dilated ureters on the glandular structure of the kidneys. Here, then, arises an important practical question. The patient has no chance of recovery without the use of the catheter. Are we to leave him to his fate, or are we to empty his bladder at certain intervals, at the risk of hastening the period of his dissolution? I have no doubt that we may, in many instances at least, obtain the good and avoid the evil by a slight modification of the treatment. Let the catheter be introduced at first so as to draw off only a portion of the contents of the bladder, and let several days elapse before it is completely emptied."

To further illustrate the effects of suddenly emptying the over-distended bladder, "let me cite a most interesting case of chronic

retention of urine, of at least three years' standing, in which no diagnosis was made until the bladder was accidentally punctured. The patient was a man of slight build naturally, but had of late become somewhat emaciated. He was seventy years old. I saw him in 1879, in consultation with a medical gentleman of New York, who gave me substantially the following account of the case.

"The patient had, three years before, noticed a marked protuberance in the hypogastric region, which he had ascribed to constipation, although he urinated with increased frequency during the day, and several times at night. The real difficulty, he thought, was the constipation, for which, and for the peculiar swelling of the belly, he had consulted several physicians. He had been under the care of the physician with whom I saw him for about twelve days. The doctor finding fluctuation, thought the disease cancerous or hydatid, and this opinion was concurred in by a senior practitioner. The attending physician feeling a little more secure on the side of hydatid cysts, on the fourth day made a puncture, two inches below the umbilicus, with a small trocar, and aspirated nine and a half pints of a light-colored fluid, some of which he took home, expecting to find in it the characteristic hooklets, but instead he found crystals of uric acid, pus, and vesical epithelium. This fluid was urine, and the trocar had entered the bladder, which had risen high above the umbilicus, and had projected so far forward as to resemble a morbid outgrowth. The catheter was not resorted to until twenty hours after this sudden aspiration of the contents of the distended bladder, then four and a half pints of bloody urine came away. Polyuria had supervened, and still the catheter was introduced but once every twelve hours, each time drawing off four and a half pints for four days, when the quantity of urine began to decrease. When I saw the patient I advised that catheterism be resorted to every six hours, but his condition was such that I gave a bad prognosis, expressing the opinion that he had but a few weeks to live. My diagnosis was chronic retention of urine from bi-lateral and supra-montanal hypertrophy of the prostate. He died in the course of a couple of weeks, and I was invited

to the autopsy. The only diseased organs were the genito-urinary, from the prostate to the kidneys."

In another case the polyuria (without glycosuria) amounted to twenty-seven pints in twenty-seven consecutive hours. Another grave consequence of the sudden evacuation of the distended bladder is cystorrhagia, which is often followed by general cystitis and pyelo-nephritis.

To completely relieve a bladder from a great accumulation of stale, putrid, purulent urine at one sitting and at the same time avert the calamities mentioned above, I suggested ten years ago the adoption of the following plan:—

Let us suppose a case in which the bladder is distended, so that its fundus rises above the level of the umbilicus. A catheter is introduced and one pint of urine is slowly drawn off. Immediately half a pint of warm borax solution—two grains to the ounce, with five minims of wintergreen essence and ten minims of glycerine to the ounce—is thrown in, then another pint of urine is allowed to escape through the catheter, and half a pint more of the borax solution is injected. There is already one pint less of fluid in this bladder, and what remains is mixed with a disinfectant. After this, for every half pint of diluted urine drawn off, half a pint of the borax solution is injected until the fluid that escapes from the catheter is perfectly clear. Once every four hours afterwards half a pint or a little more of the clearer contents is removed, until the bladder is entirely empty, which may take several days or more than a week.

Since cystitis is almost certain to be developed as a consequence of stagnation of urine, and since it is known by experience that the sudden evacuation of the distended bladder greatly aggravates this existing inflammation, which is not completely relieved by gradual evacuation, but still lingers to cause rapid decomposition of the urine, and later perhaps phosphatic stone, it is absolutely necessary to combat it vigorously and continuously in order to obtain over it some control. An absolute cure of the chronic cystitis accompanying prostatic hypertrophy has not yet been, and will not be, accomplished until it shall be possible to restore the urethro-vesical orifice to its normal state.

I have said nothing of the medicinal treatment of cystitis for reasons already given, but should like to make a few comments on its treatment by irrigation.

Vesical injections are used for the purpose of simply cleansing the bladder of decomposing phosphatic concretions, of dilating contractured bladders, or of applying certain agents to stimulate, modify, or soothe the mucous membrane.

For cleansing, warm water, with the addition of a few drops of essence of wintergreen to deodorize—but any other essence will answer the purpose—is often all that is required; the density of the water to be injected is sometimes increased by dissolving in it a small proportion of chloride of sodium, or chlorate or permanganate of potash. Phenic acid has been largely used in such cases, in solutions of various strength for the purpose of cleansing the bladder, but I fail to see in it any advantage over some of the other alcohols, and its disagreeable odor and irritating qualities render it objectionable. When the urine is highly alkaline, the cleansing fluid may be mildly acidulated with nitric, hydrochloric, or phosphoric acid. When the urine is purulent and slimy the biborate of soda or boracic acid, two grains to the ounce of warm water, seldom fails to be beneficial. After a few weeks' use of the former I have substituted the latter.

Such irrigations may be used once, or, at the most, twice daily, and not more than half a pint at each sitting, throwing in two or three ounces at a time. Much good may be expected by the prudent use of irrigations. They are, not unfrequently, used to excess, and this abuse of such an excellent remedy only results in disaster, especially when the fluid employed is of low density. In that case the mucous membrane becomes waterlogged, its epithelium is destroyed, and the cystitis is rendered utterly unmanageable.

When phosphatic concretions have formed as a result of stagnation of urine, they may be decomposed by hydrochloric acid, two minims to the ounce of warm water, slowly injected into the bladder. If this is well tolerated, it may be used once every second day, or even every day. The quantity of fluid used should rarely exceed half a pint at each sitting, but not

more than two or three ounces should be injected at a time. Another agent, which I have found very efficient in several cases, is the acetate of lead solution, one grain to the ounce of warm water, acidulated with acetic acid, as recommended by Doctor Hoskins, of Guernsey. In one case I increased the solution to three grains to the ounce, and used one pint at each sitting for thirty consecutive days, and completely freed the bladder from phosphatic concretions, which had caused much discomfort, and there was no recurrence of the trouble. In another case the strength of the solution was increased to seven grains to the ounce, and was well tolerated by the bladder. However, as a general rule, I think it safer to adhere to the weak solutions of Doctor Hoskins.

Fortunately, contracture of the bladder with diminution of its capacity is a rare complication of cystitis in prostatic hypertrophy, but when this occurs measures must be promptly taken to dilate the viscus to its normal capacity, otherwise the short life of the sufferer is rendered intolerable by the almost constant desire to urinate. A very excellent method by which this desired end may be attained, is slow, gradual hydraulic dilatation (sudden dilatation is very unsafe and generally unsuccessful). This is accomplished by injecting warm water to the point of tolerance, then, a few seconds after, by adding a very small quantity, perhaps not more than a drachm. The amount is slightly increased from day to day until, from half an ounce, which may have been the maximum quantity retained at first, in two or three weeks the capacity of the bladder is increased to eight, ten, or twelve ounces. This method was much employed many years ago by Civiale and others. I have resorted to it in a considerable number of cases with very good results.

The sulphate of quinia in solution has been greatly vaunted as a vesical irrigation to combat cystitis. In very weak solutions, that are tolerated by the bladder, it is inert, while in solutions of two, or even of one grain to the ounce, it over-stimulates and irritates the mucous membrane. Such, at least, has been my experience. The fashion was of short duration.

As a modifier of the vesical mucous membrane nitrate of silver occupies the first rank; only, however, when the urine is

of acid reaction. When the urine is persistently alkaline, this salt, even in very weak solution, is not tolerated, and should not be used. In certain cases of inordinate sensitiveness of the prostatic urethra and urethro-vesical orifice, the injection of an ounce or two of nitrate of silver solution, one grain to the ounce, is of great service, and its repetition once every third or fourth day for a couple of weeks renders catheterism much easier to the surgeon, and almost painless to the patient. In some other cases I have begun with very weak solutions (one-tenth of a grain to the ounce), and gradually increased the strength to two grains, seldom more, to the ounce. In these cases I have thrown into the bladder four ounces of the solution, making one such injection at each sitting.

I take this occasion to protest once more against the practice of filling the bladder with nitrate of silver solutions of twenty to thirty grains to the ounce, because this practice is still resorted to, though, I am glad to say, very exceptionally. From careful analysis of a number of cases in which this salt had been used in strong solutions, I am convinced that it should never, under any circumstances, be so employed in the treatment of cystitis.

To render catheterism less painful in cases of great irritability of the urethra and bladder, solutions of sundry substances are thereinto injected, or introduced into the rectum, such as morphia, atropia, hyoscyamus, and more recently cocaine hydrochlorate. I have employed it in one case with some advantage.[1]

[1] Cocaine hydrochlorate, a most valuable local anæsthetic when properly used, is now the rage, and like most newly discovered remedies is, in my belief, used too recklessly and excessively. However, experience will indicate the real capabilities of this analgesic. At present among the uses to which it is put is that of rendering the urethra insensible to the pain of catheterism. This surely will sometimes lead to evil, for it is not always wise to abolish the sensibility of the urethra, which sensibility is often so valuable a guide in difficult catheterism. Therefore it is more rational to lessen abnormal sensibility than to abolish all local sensation, and the surgeon cannot be too circumspect in the employment of substances that tend to abolish urethral pain in catheterism; he should be content to simply lessen the pain.

Cocaine hydrochlorate in solution has lately been used as a vesical injection to render lithotripsy painless. Time and further experience will settle the question of the advisability of continuing its use for that purpose.

Mr. Harrison, of Liverpool, has suggested the use of some of these alkaloids, in small pessaries or suppositories, made of cacao butter, to be placed in the urethra or bladder, by means of a specially constructed catheter. He also advises vesical injections of water at 100 to 120 degrees F. to soothe the bladder, allay spasm, etc. The other extreme is resorted to by Dr. Cazenave, of Bordeaux, who strongly advocates the free use of ice in the rectum to make catheterism possible where it had before failed.

I have resorted to both of these methods in selected cases with equally good effect.

Prostatic obstruction, as it progresses, often becomes complete, and not a drop of urine flows spontaneously. In such cases the use of the catheter is repeated four, five, or six times daily, and, however much care patients may take who catheterize themselves so frequently, they, sooner or later, suffer from subacute urethritis, and are annoyed by considerable pain in using the instrument, and by a constant purulent discharge which necessitates astringent urethral irrigations to check the discharge, and at the same time blunt the sensitiveness of the mucous membrane, and also to wash out from the prostatic sinus plugs of muco-pus which for hours give rise to a constant desire to urinate.

Owing to the irritation of the necessarily frequent catheterism, these patients are, from time to time, seized with the most painful orchitis, but it is impossible to lay aside the catheter. In some cases I have been able to relieve the pain and cut short the inflammatory process by the application of half a dozen leeches at the lower portion of the inguinal region immediately over the spermatic cord, and by the application of a small opium poultice over the leech bites, enveloping the testicle with a warm slippery-elm and tobacco poultice, besides giving a large opiate by mouth. In other cases I have been obliged to resort to multiple capillary punctures to give issue to serous accumulation before relief came. In a few cases I have found it necessary, after some weeks, to open abscesses of the testicle.

2. *c. f.—Prostatotomy and Prostatectomy.*—Before speaking of

the operations of prostatotomy and prostatectomy and of the instruments required for their performance, it may be interesting to briefly sketch the sundry devices which have led to their adoption.

Prostatic hypertrophy was looked upon by the ancients as "excrescences or carnosities" at the neck of the bladder, which demanded destruction to re-establish the course of the urine, and this destruction was attempted by the erosion of the supposed carnosities with metallic instruments.[1]

Later, when the nature of the disease was better known, the obstacle was perforated by metallic catheters or sounds. This was recommended and practised by John Hunter and Sir Everard Home, and still later by others, but finally abandoned as uncertain and dangerous.

Caustics were used for the purpose of destroying the prostatic obstruction, particularly by the followers of Ducamp.

Compression by the prolonged retention of catheters in the bladder was one of the expedients resorted to in cases of prostatic obstruction.

Dr. Physick, of Philadelphia, employed hydraulic compression and retrograde dilatation of the urethro-vesical orifice to overcome prostatic obstruction.

In 1825, J. Leroy d'Etiolles endeavored to make depression and compression by means of a rectilinear metallic sound, and afterwards by a flexible catheter, substituting a straight for the curved stylet after introduction. In both cases he really pushed back the obstacle without dilating the neck of the bladder as had been done by Dr. Physick, and his patients, for a while, urinated better. Mercier cites Meyrieux and Tanchou as having employed for this purpose an articulated stylet which could be straightened after the introduction of the catheter and kept in position for two hours. Mercier suggested his rectangular sound for depression of the prostate, and also a flexible catheter with a stout

[1] The prostate was not discovered until about the middle of the sixteenth century, and the discovery is attributed to Nicolas Massa. The first suggestion made that the neck of the bladder could be obstructed by a tumor of the prostate was by Riolan in the first half of the seventeenth century.

straight whalebone stylet, and says that he has obtained some good results with the latter, but has not retained it in the bladder more than ten minutes at a time.[1] He speaks of Nicod who, mistaking supra-montanal tumors for polypi, tore them off and thus, by this rude method, obtained some success. Riolan, in the seventeenth century, performed trachœlocystotomy for retention of urine several times with success.

Covillard (seventeenth century) after performing this operation tore away with the forceps a prostatic tumor from the urethro-vesical orifice, and in another case, during lithotomy, repeated this procedure.

Desault resorted to the same operation in one case. Sir William Blizzard is also cited as recommending and performing trachœlocystotomy for the purpose of removing prostatic obstruction.

In our own time a number of surgeons in performing lithotomy have removed, by enucleation or otherwise, prostatic tumors of various sizes. Among these surgeons may be mentioned Sir William Fergusson, Mr. Cadge, Dr. Williams, and Mr. Bickersteth. In our country this expedient has also been resorted to with success.

Leroy d'Etiolles suggested the ligature of pedunculated supra-montanal prostatic tumors.

Jacobson's lithotrite has been used as a sort of *écraseur* for the removal of such tumors.

Amussat, in performing supra-pubic lithotomy, excised a small tumor from the neck of the bladder.

The division of the obstructed urethro-vesical orifice for the purpose of re-establishing the course of the urine was not erected into a regular and systematical surgical procedure until it was formulated by Mr. Guthrie, under the name of "the division of the bar at the neck of the bladder;" but Mr. Guthrie's operation does not seem to have been generally adopted.

[1] Mr. Harrison, of Liverpool, has recently revived this method, or rather one part of it, compression, by the daily introduction of the bellied bougie (bougie-à-ventre of the French) with a view of establishing a channel in the centre of the prostatic valvule.

Leroy's and Civiale's instruments were not equal to cope successfully with prostatic obstruction.

After a careful study and analysis of the various attempts to relieve prostatic obstruction that had been made by the surgeons who preceded him, Dr. Mercier was led to devise an instrument to excise a portion of the obstruction, and thus make a channel through which the urine could be more freely expelled from the bladder. His first instrument was made in 1837 by Charrière. In 1838 he tried it several times on the cadaver; in March, 1839, exhibited it to the Society of Anatomy, and on the tenth of December in the same year described it in a letter addressed to the Academy of Medicine. He has since devised three new instruments, one for excision and two for incision.

Mercier gave no names to his instruments, except that of incisor and excisor, and to his operations that of incision and excision of urethro-vesical valvules.

I have named the instruments prostatotome and prostatectome, and the operations prostatotomy and prostatectomy.

Internal prostatotomy and prostatectomy are executed by means of special instruments, not unlike lithotribes in appearance, and introduced in a similar way.

External prostatotomy and prostatectomy, the suggestion of which can be traced to Mr. Guthrie, are preceded by external perineal urethrotomy of the membranous portion. The former is done with a special prostatome, or simply with a straight probe-pointed bistoury, or a concealed cystotome; the latter with a special prostatectome or, as in the case of a pedunculated tumor, with a small wire écraseur, or in an isolated intra-mural tumor, by incision and enucleation.

About fifteen years ago, an Italian surgeon, Dr. Bottini, proposed and practised galvano-cautery for the purpose of avoiding hemorrhage in the destruction of prostatic obstruction, and says that he has obtained good results. Hemorrhage is seldom formidable in prostatotomy or prostatectomy, and is generally soon controlled; therefore the utility of this more complicated operation by electro-cautery may well be questioned.

I need not describe Mercier's instruments, for this has already

been done by himself and others. On account of the difficulty which I have experienced with Mercier's prostatectome in surmounting from behind forward the urethro-vesical obstruction with the male blade, I devised a prostatectome, of which the following is a brief description :—

This instrument resembles an ordinary modern lithotribe, and its mechanism is very simple. It consists of a male and a female blade, the one sliding into the other. The male blade is hollow, its sides are vertical and its superior portion a little rounded. It is welded to a tubular shaft, in the centre of which is a long needle worked by a button, at the proximal extremity of the handle. The object of this needle is to transfix the prostatic valvule or tumor, and hold it in position from the moment it is seized till it is cut and extracted, the cavities in the two blades rendering extraction certain. The inclination of the male blade is such that it will ride quite easily over the valvule as a preparatory step to prehension, whereas in Mercier's instrument the same blade is bent so abruptly that it is with the greatest difficulty carried over the valvule, and I have found it impossible in some instances. Again, what in Mercier's *exciseur à hameçon* is a cutting edge, in mine is a *flat* edge, so that it really acts as an *écraseur*. This instrument will cut or punch out a segment of the valvule three by nine millimetres. The beak is hollow to receive the piece of valvule, in part at least, and has a small orifice at its extremity, which serves the double purpose of allowing a little water to escape and making sure that the bladder has been reached, and of forcing out any blood oozing from the cut, which might otherwise soon coagulate, fill the cavity, and interfere with the proper lodgement of the segment of valvule. The shaft of the female blade is cut sloping for four centimetres to its junction with the beak, so as to give more depth to the male blade. The handle is copied from Collin's lithotribe handle. The instrument is easily dismounted for cleansing by first unscrewing the button which serves to work the needle. Marks one centimetre apart on both surfaces of the screw apparatus indicate the extent of separation of the

blades during seizure of the valvule, and consequently its thickness.

To seize and punch out a piece of the valvule, the instrument is introduced locked, the beak is reversed as soon as it has entered the bladder, and the screw turned from right to left, so as to open the blades or jaws, when the male blade will pass over the valvule and drop in front of it into the prostatic sinus. The instrument still locked is moved to and fro that the operator may know that it is in its proper position, then the needle is thrust through the valvule until its point has entered a shallow pit in the posterior wall within the female blade. The screw is now turned from right to left until there is a slight resistance. The operator casting his eye upon the handle at once ascertains the thickness of the valvule which he is about to cut. A few slow turns of the screw which drive the male blade home will complete the section or *écrasement*, with a peculiar crunching sensation, such as is never experienced in any other operation. The instrument, holding the segment in its closed jaws, is pushed into the bladder and then slowly withdrawn, and a full dose of quinine is given. In the course of half an hour the patient is asked to urinate; failing, his bladder is emptied by means of a catheter. Contrary to Dr. Mercier's injunctions, I have retained a catheter in the bladder for twenty-four hours in some of my cases.

The after-treatment consists in the introduction of an instrument to make dilatation and depression of the urethro-vesical orifice once every five days to retard the process of cicatrization.

RESULTS.

Dr. Mercier told me seven years ago that he had performed these operations about four hundred times, and that of all the complications hemorrhage had given him the least trouble, and that his results had been extraordinarily good. Other surgeons, however, have not been so fortunate; for two cases, by incision, operated upon by Dr. Laugier in 1863, at the Hôtel Dieu of Paris, died apparently of pyæmia; one in sixteen days, the other

in thirty-three days, the latter having advanced renal disease. In 1863 and 1865, Dr. Demarquay operated upon three cases by excision. Two cases were successful, and the third died on the fifth day from the consequences of hemorrhage and intra-pelvic suppuration. Here, then, is one case of fatal hemorrhage in five operations. This would be a large proportion if the accident occurred once in every five cases, but it does not. Among Dr. Mercier's published cases is one only of profuse hemorrhage, which it was difficult to control, but the patient eventually made a good recovery. Other French surgeons have performed the operation, but I have not their statistics. The effect of the operations of incision or excision may be transitory or permanent: transitory, when the difficulty in urination is reproduced; permanent, when it is always possible for the patient to empty his bladder without the aid of a catheter. Mercier relates a number of cases in which the patients were able to empty the bladder from the moment of the operation, and continued to do so indefinitely. In others, however, there was only an improvement in micturition, but this improvement was progressive. In another class of cases there was a considerable amount of residuary urine, but ordinary catheterism, impracticable before, was rendered possible and easy by the operation.[1] Recurrence of the troubles in urination has been noticed in some cases, and in these Dr. Mercier considers it proper to repeat the operation even years after the first operation.

I have had recourse to cutting operations for the relief of urethro-vesical obstruction in nine cases. The ages of the patients were as follows: one was twenty-six, one forty-eight, two were fifty-nine, one sixty-seven, one sixty-eight, two were seventy, and one seventy-six years of age.[2]

[1] Some years ago I saw a patient upon whom Dr. Mercier had operated twenty years before, and he was able to sound himself easily.

[2] My first operation of internal prostatotomy was performed on the 4th of October, 1878, and my first external prostatotomy was performed on the 27th of April, 1880. My first internal prostatectomy was on the 15th of June, 1880, and my first external prostatectomy was on the 20th of July, 1880.

The obstruction was of two years' standing in three cases, of four years in two cases, of seven years in one case, of eight years in one case, and of ten years in two cases.

Of the nine operations, two were of internal and two external prostatotomy; and three of internal and two of external prostatectomy. Eight of these patients recovered and one died, the cause of death being advanced pyelo-nephritis. There was a considerable hemorrhage in one case only, and this bleeding was controlled in a short time by cold irrigations, and the retention of a catheter.

In one case the patient was able, after the operation, to catheterize himself easily, while before, ordinary catheterism was very difficult, and sometimes impossible. This was the only advantage gained by the operation. The patient died three years after at the age of seventy-nine.

In one case the result was not as satisfactory as I had expected, since the patient was only at times able to empty his bladder spontaneously. Of the remaining six cases, five patients were after a while able to empty the bladder spontaneously. The sixth still retained a small amount of urine when he left me. The daily use of the catheter was advised in all these cases for the double purpose of irrigating the bladder and of maintaining the patency of the cut made in the urethro-vesical barrier.

In three of these six cases there was in the course of a couple of years a recurrence of the urethro-vesical obstruction indicating the necessity of a second operation, but it was not performed. Two of these patients have since died, one three years, the other five years after the operation.

INDICATIONS AND CONTRA-INDICATIONS.

Prostatotomy and prostatectomy, in my belief, should be reserved for cases of crescentic valvular obstructions, and bars of not more than a centimetre in thickness. I would give my preference to external prostatotomy over the internal operation in these cases, although I have had recourse to external prostatotomy and prostatectomy in only four of my nine cases.

As a general rule it is best to perform the operation in the early period of the existence of obstruction to urination, but under certain circumstances it is justifiable to operate upon cases where the obstruction has been of long standing, for instance, when ordinary catheterism becomes very difficult or impossible.

Advanced renal disease is a contra-indication. Dr. Mercier has operated successfully upon cases where stagnation of urine had existed seven and nine years.

The coexistence of stone is not a contra-indication; nor is age, some of Mercier's patients were 74, 76, and 78; one of mine was 76.

Predunculated tumors (which are rare) should be removed by external operation, and if large, with the aid of a small wire écraseur, if small, by clamping them with Mercier's last prostatotome, and cutting through the pedicle. Intra-mural tumors also require external section followed by enucleation. This method has been eminently successful.

I doubt if anything can be gained by excising a portion of a large sessile supra-montanal growth, and am disposed to think that a very free central incision would be more serviceable.

DISCUSSION.

Dr. S. W. Gross, of Philadelphia, Pennsylvania.

I think in regard to the general measures for relieving retention of urine from enlargement of the prostate gland, the Fellows of the Association will be in entire accord with Dr. Gouley. His remarks are eminently judicious, especially in regard to the treatment of the bladder when the patient enters upon what is termed his catheter life. We are all aware of the fact that if the bladder be entirely emptied and the process be repeated from time to time, death is liable to ensue in a short time from surgical kidney. I think that his practice of drawing off a portion of the urine and throwing in a borated solution is most wise.

The instrument which I hold in my hand, and which I think is the instrument for measuring the enlargement of the median prostatic outgrowth strikes me as being excellent for determining the amount of thickening and for determining the nature of the operation for its relief.

In this particular direction, surgery is capable of making great advances, that is in the treatment of ischuria from median enlargement of the median prostate. I myself believe, and I think that Dr. Gouley stated the same, that it is a proper procedure to make a median incision in the perineum and through the obstructing body. Harrison, although he did not originate the operation, is the reviver of it. The first case reported by Mr. Harrison was that of a man in whom he opened the membranous portion of the urethra through the perineum. The obstruction was divided partly by incision and partly by divulsion with the fingers, and the edges kept apart by a large tube through which a smaller tube was passed to conduct off the urine. The patient was able to go about in ten days. On the withdrawal of the large tube a bougie was passed to keep the edges apart until the wound has closed. All obstacles to micturition were overcome, the urine was passed every four hours and the bladder completely emptied. At the end of six months the patient had a paralytic seizure, and there was no necessity for resorting to the catheter. The case was an unfavorable one for operation, but the result was brilliant. Through a small perineal wound the nature of the obstruction was determined and the incision was made with a degree of accuracy and safety which cannot be attained with Mercier's cutting instrument.

Only a short time ago Bottini, of Pavia, operated with the thermo-galvanic cautery applied for forty-five seconds. A catheter was retained for four days, and the bladder was subsequently emptied every six hours. The first natural emission of urine took place on the 22d day. At the end of three months micturition was normal, and at the expiration of three months and a half more the patient was restored to health. As in the operation of Mercier, there is no certainty in operating with the thermo-electric cautery, and if we consider the great tendency of cicatricial tissue after burns to undergo undue contraction, it is a matter of grave doubt whether the improvement will be permanent.

I believe that the operation revived by Mr. Harrison is the better one, and the one entitled to extended trial.

As soon as Mr. Harrison reported his case I asked him to send me the tubes which he had used. As you see the large tube is nothing more than the English tube for draining the wound after lithotomy.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

The hour is so late that it is not advisable to make any extended remarks, but most of us will concur in the judicious remarks of Dr. Gouley in the general treatment of these cases, and especially the stress that he has laid on the use of metal and hard instruments. These are of course liable to give rise to injury. The early use of the catheter in cases of obstructed urination from enlarged prostate is a very important point which the author of the paper has dwelt upon. I do not know that anything is to be added to that subject. Certainly it should be done before the damage, which will invariably follow in the bladder, takes place. It should anticipate and prevent the evils. It is useful more as a preventative that as a measure of relief.

There is another point which he touched upon, and that is frequent urination in over-distended bladder. This is frequently overlooked. When a patient is said to have dribbling of urine, it should be looked upon as a sign of retention and not of incontinence. We not infrequently hear of cases where the obstruction is due not to enlarged prostate but to other causes, such as typhoid fever, where the bladder may become enormously distended. Where there is dribbling of urine it is supposed that the catheter is not necessary. It is an indication for exploration of the bladder.

In reference to the quantity of water used in washing out the bladder, it is stated that three or four ounces could be injected. Sir Henry Thompson urges that not more than one ounce be thrown in. I do not know that it would make a great deal of difference if it were cautiously and slowly injected. I fully concur with the opinion expressed in regard to the injudicious use of injections rapidly thrown into the bladder, leading to rapid distension. It has occurred to me that this process of hydraulic distension might be very dangerous in those cases where great contraction has taken place from obstruction, if done where there was reason to think that there was involvement of the kidneys; where, for instance, there was surgical kidney. We might have the same condition of shock which follows the rapid evacuation of a large quantity of urine.

There are many other points suggested by this paper which we have not time to discuss.

Dr. Edward M. Moore, of Rochester, New York.

I must confess to being indebted to Dr. Gouley for his exhaustive paper. Few things more important come into the hands of the practical surgeon. I wish to say that I regard the point already alluded to as the most important point in all the paper, that is, the danger of rapidly emptying the over-distended bladder. I think that the ordinary profession as we find its members practising throughout the country are not aware that this practice is attended almost invariably with severe symptoms and even with death in a short time. The injections of boracic acid we should indorse with great heartiness.

As regards the operation, as a matter of course it is done but rarely. When I was in Copenhagen, I had a conversation with Mr. Harrison, and I think he cited eight cases, all of which he claimed to be successful. In all the cases he resorted to simple incision and in no case to excision of any portion of the prostate gland, relying upon the fact that if the part was kept distended, the process would be perfect in a few weeks and then there would be little or no difficulty. The instrument has been shown here to-day.

There is another important point which Dr. Gouley has not mentioned, and that is with reference to the treatment of these cases after we have evacuated the bladder successfully. Suppose we have gone through with the process of emptying it step by step, until we have passed by that period of acute cystitis which is so alarming. In passing let me state that the case referred to in which twenty-seven pints of urine were drawn from the bladder in twenty-seven consecutive hours was my own case. I was called to see the case and drew off a pint of urine, then stopped intending in four hours to draw off more. In an hour, the pain again became severe and I was obliged to pass the catheter and again drew off a pint. This was repeated for twenty-seven consecutive hours, drawing off a pint an hour. This polyuria seemed to come from the fact that the valves at the ureter were useless and taking off the strain from the kidney we at once had this enormous secretion. When we take the strain off the bladder, we have the same secretion of mucus. Supposing then that the bladder is emptied, and suppose that the patient is able to pass urine himself. The natural irritability impels

him to micturate every hour or half hour. There comes in here a method which I consider of a good deal of importance. I am sure that I have succeeded in many cases of securing relief by what I call the moral method. I advise the patient to resist his inclinations. We all know that when there is an instinctive tendency to pass water, if it is resisted, the desire passes away. If the patient resists the desire, a little time is gained. Then he can yield to the second impulse. In this way we get nature's method of distending the bladder; I go on until I reach an interval of four hours which I advise the patient to adopt as the time for emptying the bladder. After the patient has acquired the ability to pass water my custom is to have the catheter passed after he has passed all that he has able to and empty the bladder perfectly once a day. This comes to be less and less in many cases.

I once had to pass a catheter for enlarged prostate in a man 97 years of age. Finally the use of the catheter was dispensed with, the irritability remaining. He lived to be 105 years old. I think that is the oldest case I ever had anything to do with.

Dr. Moses Gunn, of Chicago, Illinois.

I wish to make a remark in corroboration of Dr. Moore's statement. It has been my practice during the last five years I have employed a similar method directing the patient to go a little longer each time and finally hit upon the same interval as Dr. Moore, four hours. I have found that this distends the bladder and allows accumulation to occur, preventing the chronic contraction which so often supervenes where the patient is unable to retain the urine for any length of time. I have found it a most admirable method.

Dr. Gouley. I have labored under the disadvantage of not having had time to refer to the cases on which I have operated. I would ask Dr. Gross the date of Harrison's operation. I have his paper, but I do not recall the date. (Dr. Gross had left the room.) Of this I am sure, however, that I antedated him. In fact I called Harrison's attention to the subject in New York, showed him my instruments, and my operation, and gave him all Mercier's ideas. He has published an essay, but never mentions my name. I am quite confident that I am the first man that ever performed external prostatotomy or prostatectomy. Mercier gave me credit for it in a private letter. Guthrie recommended it, but it is uncertain

whether he ever performed the operation. Mercier had never done it. I am not aware that any one since Mercier had proposed the operation of prostatotomy or prostatectomy, least of all to name it.

It was not until after I had studied the subject for a considerable time and experimented upon the cadaver that I dared to undertake the operation of internal prostatotomy. That was my first case. Then I further experimented on the cadaver, and then operated on my second case, performing the external operation. I left no instrument in. I said that to Harrison. I was going to say that Harrison was present at the operation, but I am not sure of that without referring to my notes. The internal operation was performed in October, 1878, the external one in 1880, in March. The third operation was in January, 1881. In that case I left a large sized rubber tube in the bladder.

In reference to a remark made by Dr. Prewitt, I would say that the idea that dribbling of urine in elderly men was due to retention and not incontinence, was first elucidated by Mercier, and as I stated in the first part of the paper, everything that we know comes from Mercier. He has exhausted the subject. Everything now is a matter of detail. I am sorry that Thompson does not give Mercier credit for that idea which is down in his first book of 1841. He was working in 1836. This idea is given beautifully in his book before the time of Sir Henry Thompson.

In regard to the irrigation of the bladder there can be no absolute rule. Some patients will not tolerate more than half an ounce of water made dense—pure water, still less. The amount is gradually increased in these cases of contracture with diminished capacity. I shall not dwell upon this point, because I have recently read a paper on contracture of the bladder. In some cases I have literally had to increase by drops, and it has taken three or four weeks to obtain the required amount of dilatation, and prevent the patient from experiencing the bad effects of over-distension. When the bladder is in a state of irritation, intolerant of urine and the desire to urinate being obeyed every five or ten minutes, it will give rise to serious symptoms, and that condition known as contracture with diminished capacity. The suggestion of Dr. Moore I have already acted upon, and I suppose that it has occurred to others. I have directed my patients not to obey these calls, but to resist them as long as possible. It is rare that success will be attained by this. It is only partial. I have tried it many times, in fact in almost every case.

This is not enough, and I am compelled to resort to gradual dilatation of the cavity, and the urgent symptoms disappear. The catheter is introduced to completely empty the bladder and prevent decomposition.

Dr. Moore has interested me very much in his citation of the case of the man 97 years old. A point that I should like to clear up is as to the time that these symptoms came on.

Dr. Moore. I cannot say.

Dr. Gouley. The man may have had enlargement of the prostate for a number of years. This point has been investigated by pathologists, notably Messer, who found that if a man is going to have enlarged prostate, he generally has it before the age of seventy. The disease begins between the ages of fifty-five and seventy. If after the age of seventy a man has had no difficulty, he probably is not going to have any. I have seen many old people who had not the slightest symptom of prostatic enlargement.

THE CULTURE OF SURGICAL BACTERIA.

COMMUNICATED ON INVITATION BY H. C. ERNST, M.D.,
OF BOSTON, MASSACHUSETTS.

THE recent work upon bacteria which has been done in Germany is of special interest to surgeons, because a portion of it has been devoted to a study of the bacteria concerned in certain acute surgical diseases. My work has been in the line of that of Rosenbach, who has found that in the pus from acute and chronic diseases, such as erysipelas, pyæmia, septicæmia, and so on, there are several forms of micrococci, which, by the older methods of fluid cultivation, were indistinguishable. They behave in exactly the same way to staining reagents, are of nearly the same size, and in a fluid culture present the same characteristics. The method introduced by Koch of employing a solid culture medium has been applied by Rosenbach to the investigation of these micrococci, and it has enabled him to differentiate these organisms.

In the course of some work which I have been enabled to undertake through the kindness of Dr. Warren, I have made a number of colonies of these organisms, and it seemed to me that it would be interesting to show what they were. I have under the microscope a culture from a perinephritic abscess which occurred in Dr. Warren's service in the Massachusetts General Hospital. The pus was transferred to a sterilized tube, and a culture was obtained showing the characters of that I have in this tube. I shall send around Rosenbach's book in which is a plate showing two cultures, the one the micrococcus pyogenis aurens, and the other the micrococcus pyogenis albus. They present a yellow and white appearance respectively. The culture medium which is used is the fleisch-peptone agar-agar.

These tubes show almost identically the appearances seen in the plate.

I brought another tube, meaning to show the two growing together. Day before yesterday this showed very well, but during the past twenty-four hours the yellow coccus seems to have overgrown its rival.

The method of differentiating these different forms is shown in this plate. The method is to take a fleisch-peptone-gelatine tube, plunge a platinum needle into the fluid to be experimented with, and transfer this into the nutrient gelatine. These different micro-organisms will show themselves by different colors. This is the method used in Berlin almost exclusively, for separating the various forms of bacteria.

Another case was of a peculiar growth over the knee, the nearest diagnosis that could be made being to call it a "papilloderm." The growth was of a dark red color, and about six inches long by four inches wide. The patient was etherized, and a cultivation made from beneath the skin. The colony which I first obtained was the white growth which is being passed around, and I also obtained this peculiar color which I have not seen described. I have called it the citreous, although it is not exactly a citron color. This micrococcus is exactly the same as the other, with the exception of the difference in color.

Of the other organisms that are of particular interest on this occasion, there is the special one of erysipelas. This colony is a cultivation of the second generation from a case of erysipelas. The micrococcus is very similar under the microscope and in its behavior to staining reagents to the others, but there is quite a difference in the appearance of this colony and those in the preceding tubes.

In connection with this work, I should like to exhibit a little picture showing the difference under cultivation between Koch's comma bacillus of cholera and the comma bacillus of cholera nostras. By experiments it has been found that the bacillus of Asiatic cholera will liquefy the gelatine in a gelatine tube to a certain point which is shown here, while the bacillus of cholera nostras will liquefy it to this point in the same length of time.

It has been said that Koch asserts that this peculiar liquefaction was a mark distinguishing the bacillus of Asiatic cholera from everything else. This is not so. It only differentiates it from the bacillus of cholera nostras.

DISCUSSION.

Dr. J. Collins Warren, of Boston, Massachusetts.

I should like to say a few words supplementary to what Dr. Ernst has said. The question of the etiology of traumatic inflammation, of suppuration, of erysipelas, and other affections, has excited much interest for many years. It seems probable that we shall be able at last to demonstrate the actual connection between germs and inflammation. Rosenbach has pointed out a simple way by which we may do this.

When I went on duty at the Massachusetts General Hospital in March, I asked Dr. Ernst if he would not undertake to make some cultures from cases in the wards. The first case was one of perinephritic abscess. The abscess was entirely excluded from the outside air, and it was a good case in which to test the presence of germs. It was opened under antiseptic precautions, a tube was inoculated, and the coccus which you have seen was cultured. Ten days later the patient died of pyæmia, and miliary abscesses were found in the heart and kidney. Microscopical examination showed the presence of the same coccus.

Another case has been referred to, that of a peculiar growth over the knee of a young girl, which dermatologists regarded as some form of papilloderm, and which I thought might possibly contain the bacillus of tubercle. Dr. Ernst inoculated several tubes from this neoplasm, and we found this citron-colored growth. Microscopic examination of the growth which was removed showed it to be an epithelioma. Whether there was any relation between the tumor and the peculiar germ not hitherto described, I shall not undertake to say. It shows the interesting direction of such experiments.

Another notable result has been the adoption of cotton which constitutes the corks of these tubes as an aseptic dressing. Dr.

Ernst suggested the use of this sterilized cotton for dressings. A temperature of 150° C., continued for an hour, is sufficient to sterilize large quantities of cotton. We have only just begun the use of this dressing, and it is a little premature to bring it before this Society. Last week I tried it on a case in which a tumor was removed from the arm with a portion of the muscle. We had union by first intention.

Dr. S. W. Gross, of Philadelphia, Pennsylvania.

I move that the thanks of the Association be tendered Dr. Ernst for his interesting and valuable demonstration. I would suggest, in the event of his publishing a paper within a short period, that as he has made these remarks before the Association, the paper be transmitted to the Association for publication in its Transactions.

Dr. Ernst. I am much obliged to the Association. In regard to the question of publication, I wish it to be distinctly understood that I show merely the cultures to be found in Rosenbach's book. I claim nothing but that they answer to his description.

I intend to carry these cultures on, and expect to perform a large number of inoculation experiments. I shall be delighted to place any publication that I may make at the disposal of the Association, but I shall not make it for a year at least. The field of scientific investigation lies open before us in this direction, and I feel strongly that our work in this country has not done us credit in this branch of science. There has been too much publication and too little work. If the Association wish what I may have in eight or ten months, I shall be glad to give it.

Dr. Gross. As inoculation experiments are to be made during the coming months, I move that Dr. Ernst be requested, if it meets with his pleasure, to attend the next meeting of this Association, and read a paper upon the results of his investigations, which will appear in the volume of Transactions. The importance of this subject is such that I need not dwell upon it.

AN EXPERIMENTAL AND CLINICAL STUDY OF AIR-EMBOLISM.

By N. SENN, M. D.,
OF MILWAUKEE, WISCONSIN.

Sudden and unexpected death during an operation is a calamity which never fails to strike terror to the heart of the boldest surgeon. Although death is a frequent and familiar visitor wherever human beings exist, nevertheless its sudden and unforeseen advent conveys with it more than the usual halo of sadness, and when such a scene transpires in the operating room it leaves impressions which neither time nor space can erase. Disasters of this kind come without warning and usually at a time when least expected. The surgeon who has the misfortune to meet with such an accident is not only destined to burden his memory with the unpleasant remembrances of the incident for the remainder of his days, but in addition he is often made an unjust object of reproach by those who are unable to appreciate the nature of the case. His conscience may be relieved by a favorable verdict regarding his conduct and management of the case before the only competent tribunal, composed of his colleagues and medical press, but that most uncertain of all things, public opinion, will, in all probability, be arrayed against him. In one sad moment the object of his ambition, the ultimate aim of his lifework has suffered irreparable loss. The surgeon who seeks to maintain and advance the interests of his profession as well as his own reputation should familiarize himself with all the causes and conditions which may precipitate such an unhappy result, with a view to adopt and apply timely prophylactic measures. Believing that it is good practice to prepare for war in time of peace, I intend on this occasion to call your attention to one of the most dreaded and, I may add,

one of the most uncontrollable causes of sudden death; I allude to air-embolism.

After consideration of the subject from an historical, experimental, and clinical standpoint, I shall endeavor to point out the conditions remote and direct which give rise to this accident. The different explanations of the immediate cause of death will be discussed and, finally, I shall offer some practical suggestions relating to the prophylactic and therapeutic measures.

By air-embolism, I understand the presence of free atmospheric air within the vascular system during life and in sufficient quantity to give rise to symptoms of obstruction. It is a true embolism inasmuch as the location of the volume of air which constitutes the embolus is always some distance from its point of entrance. The presence of air in a vessel offers the same mechanical obstruction to the flow of blood as a solid substance, and gives rise to the same disturbance of circulation in the tissues supplied by the vessel. An air-embolus differs from an ordinary embolus that, when once introduced into the circulation, it is capable of being broken up or divided by the blood current and the action of the heart, and on this account usually becomes the source of multiple emboli. Pathologically it also differs from a solid embolus, inasmuch as it is more likely to be removed by absorption and is less liable to be followed by thrombosis. Air-embolism is always due to the introduction of atmospheric air into a wounded or injured vein, and, in contra-distinction to the ordinary form of embolism, it is primarily most always formed in the right side of the heart and in the venous system. With a view to study the immediate effects of the presence of a considerable amount of air on the heart and the vessels, it becomes necessary to allude to the experiments which have been made where embolism was artificially produced by the introduction of solid substances into the circulation.

I. *On the Immediate Cause of Death by rapidly Fatal Embolism.*

The most interesting experiments on embolism were made by Virchow, in 1847, and by Panum, in 1854–1855, and although

the conclusions of these experimenters are somewhat at variance, our present knowledge on this subject is based upon the conjoined labors of these distinguished writers.

Virchow was convinced that his experiments were conclusive in showing that complete embolism of the pulmonary artery would invariably prove fatal in a short time, while partial obliteration of this vessel produces either no symptoms at all or only temporary dyspnœa, restlessness, and a sense of oppression. He gives the following explanation to account for the immediate cause of death in cases of complete obstruction in the pulmonary artery by a large embolus.[1] "The first effect of the pulmonary ischæmia is the interruption of the supply of oxygenated blood to the coronary arteries of the heart and the arteries of the body, as well as the stasis of the venous blood in the right side of the heart, the coronary veins, and the veins throughout the body. These conditions result in the arrest of the heart's action in the diastole, the tetanic contractions of the voluntary muscles, the retardation of respiration, the dilatation of the pupils, the protrusion of the eyeballs, etc., and very soon complete death."

Panum, by a series of very ingenious experiments, disproved the assertions of Virchow, that a lack of arterial blood in the coronary arteries produces instantaneous arrest of the heart's action.[2] In a rabbit, where the ventricular contractions had ceased for fully fifteen minutes and where only the right auricle continued to pulsate, he injected a warm black mass, composed of tallow, wax, and soot, into the aorta for the purpose of studying more accurately the anatomical relations of the coronary arteries. The injection penetrated the smallest vessels. The right auricle continued its rhythmical movements for three and a half hours after the injection was made, the heart and lungs having been removed from the body. In a second experiment he divided both pneumogastric nerves in a dog, and then opened the chest and the pericardium and passed a double ligature underneath the innominate artery; after tying the upper ligature

[1] Gesammelte Abhandlungen, p. 297.

[2] Experimentelle Beiträge zur Lehre von der Embolie. Virchow's Archiv, xxv. p. 308.

and drawing the lower tight, the artery was opened between them and a silver tube introduced and secured in the vessel with the proximal ligature. The silver tube was connected with a glass tube by interposing a piece of rubber tubing. To the distal end of the glass tube another piece of rubber was attached and all tubes filled with oil, which was kept from leaving the tubes by a clamp. The aorta was compressed with a spring forceps above the origin of the innominate artery and the same black mass was injected through the tubes into the aorta. The oil and black mass entered the vessel, and after closing the aortic valves filled the coronary arteries, which were found completely blocked with the foreign substance. The movements of the heart were carefully observed before, during, and after the injection was made. Before the injection, the contractions were regular, 80–90 per minute; during the injection, on account of the higher temperature of the injection (45° C.), the contractions became more rapid, and in the left side of the heart, which was distended with blood, they were less forcible, an occurrence which could be readily accounted for by the mechanical obstruction to the outflow from the left ventricle. All the chambers of the heart continued to contract for five minutes after the injection was complete. Six minutes after the injection was made, the contractions of the left auricle ceased. The movements of the ventricles were feeble, but could be plainly seen. The rhythm of the ventricular contractions grew slower than the contractions of the right auricle, and at the same time were less regular. After twenty-five minutes the right auricle pulsated 48, the ventricles 24 times per minute. Five minutes later the pulsations of the auricle and ventricles were the same in frequency. Forty minutes after the injection the ventricles contracted 24 times per minute to 8 auricular pulsations. Fifty minutes after the injection the ventricles made 23 rhythmical movements to 3 of the auricles. After one hour the movements of the heart again became regular, inasmuch as the auricles and ventricles pulsated 13 times per minute, and in such a manner that the movements of the ventricles followed immediately after the contraction of the auricles and were followed by a long dias-

tolic pause. Seventy-five minutes after the injection the movements of the left ventricle ceased, while the right half of the heart contracted regularly 8 times per minute. Two minutes later the ventricle contracted only twice to 8 movements of the auricle. Ninety minutes after the injection, the right ventricle ceased to beat, while the right auricle continued to contract for 6 hours and 10 minutes after the injection, making toward the last only 1 movement per minute. After all pulsations had ceased for a while they were renewed by blowing upon the heart. These contractions continued for seven and a half hours after the injection, and even after they had ceased for a second time they were again excited by mechanical irritation. During this observation the heart was kept at a temperature of 12.5 to 13° C under a glass bell in which the air was saturated with moisture. An examination of the heart showed that the coronary arteries were completely distended with the black mass, and that the capillary vessels and coronary veins were blocked with oil. A few drops of oil, but nothing of the black mass were found in the right auricle. The aorta near the heart was filled with the injection material, and the aortic valves were so completely closed that nothing had penetrated into the ventricle. In two other instances the coronary arteries were made impermeable in a similar manner, and the contractions of the heart were temporarily arrested by electric irritation of the pneumogastric nerves. This experiment was repeated in both animals more than 20 times, and always with the same uniform result. If the electric stimulation was continued after the heart had ceased to contract, the movements were again excited, but this always required a continuation of the current for at least a minute. If the electrodes were removed after the heart ceased to act, it required twenty seconds before the contractions were re-established, the movements being always more rapid than before the irritation was applied. From these experiments we are forced to conclude that embolism of the coronary arteries is insufficient to produce instantaneous arrest of the heart's action. Virchow quotes Erichsen as having observed prompt cessation of the movements of the heart after ligation of the coronary arteries, but Panum doubts the possi-

bility of performing this operation upon the heart of a living animal.

The same observer studied embolism of the pulmonary artery by injecting an emulsion of gum arabic, in which were suspended small pellets of black wax, into the jugular vein of a medium-sized dog. Eight c. ctm. of the emulsion were injected. All signs of life and all reflex movements ceased three minutes after the injection. After death the large vessels were tied, in order to ascertain the exact quantity of blood contained in each side of the heart. The right side contained 112.35 grms. of dark-colored blood, the left contained only 6.45 grms. In another experiment he injected coarsely powdered charcoal in suspension into the jugular vein of a dog. The time which elapsed between the injection and cessation of life was longer than in the preceding case, consequently the left heart contained a larger amount of blood, although the quantity was small when compared with that of the right side of the heart. In all cases of death resulting from embolism of the pulmonary artery, the amount of blood found in the left side of the heart is proportionate to the completeness of the obstruction in the pulmonary artery. The left side of the heart is never found completely empty, as the labored respiratory movements will force the blood, which is present in the pulmonary vein and its branches, into the left side of the heart. If instead of using small emboli large plugs are injected, as was done by Virchow, the blood contained on the distal side of the obstruction will pass through the pulmonary circulation and reach the left side of the heart, consequently in such cases more blood will be found in the left ventricle. Panum asserts also that the cessation of the heart's action does not invariably take place so early that it can be considered as the primary and direct cause of death. As a rule, he found the heart pulsating after the death struggle had been initiated from arrest of innervation from the cerebro-spinal centre. In some instances the heart continued to pulsate after all signs of animal life emanating from the brain and spinal cord had ceased. Shortly after respiration was arrested the heart did cease to pulsate, and, as Virchow has stated, in the diastole.

According to Panum, the cessation or continuation of the heart's action exerts no influence for good or evil in cases of extensive embolism. He claims that if the cessation of the heart's action takes place as one of the first effects after embolism of the pulmonary artery, as was noted in Virchow's first case, it must be regarded, under certain circumstances, as being the result of irritation of the pneumogastric nerves, so much more so, as the heart in the case referred to again began to pulsate after the thorax was opened. As a rule, the heart's action is arrested by distension of the right ventricle.

Other observations tend to show that the distension of the right ventricle is the cause. The excess of carbonic acid gas and the diminished supply of oxygen must also be taken into account. Other experiments have demonstrated that carbonic acid, in concentrated form, injected into the heart after its removal from the chest readily leads to diastolic paralysis, and that the organ commences to beat again when exposed to air. The arrest of the heart's action is due to mechanical dilatation and the presence of an excess of carbonic acid. The first and most constant symptom resulting from sudden and extensive embolism is a high degree of anæmia in all visible parts of the body. On post-mortem examination the white substance of the brain is completely bloodless, especially if small and numerous emboli have been injected. This general anæmia is followed by tetanic stretching of all extremities, involuntary discharges, and deep, convulsive, inspiratory movements. Ligature of both carotid arteries does not produce such an intense ischæmia of the brain. If the vertebral arteries are ligated at the same time, the tightening of the ligature of the second vertebral artery produces syncope and convulsions, but the symptoms are less intense than after sudden, fatal embolism of the pulmonary artery. Panum also induced cerebral anæmia by injecting black pellets of wax, suspended in an emulsion, into the crural artery of a dog, throwing the injection in a central direction through a catheter which had been passed into the artery near the heart, producing thus multiple embolism in all of the smaller arteries. The animal lost only a few drops of blood, and no air entered.

The animal was taken immediately with tetanic convulsions, involuntary discharges, and all organs accessible to the eye presented an extremely anæmic appearance. All reflex symptoms were arrested after one to two minutes. Two other experiments were followed by the same results. In all cases the small wax pellets were found in large number in the small vessels of the brain, as well as in all other parts of the body. In four other dogs cerebral embolism was avoided by introducing the catheter only as high as the ribs, and by injecting slowly. During the injection a peculiar tremor was observed, which affected the muscles of the lower extremities; this, however, soon ceased, and gave way to complete paralysis of both motion and sensation, as well as complete arrest of all reflex movements. One of the animals survived the experiment 22 hours, the second 9½ hours, the third 6 hours, and the fourth 5 hours. The small vessels of the spinal cord were found obstructed by the small wax pellets, the vessels between the emboli and the heart were much dilated, and showed many small extravasations. The spinal cord was the seat of red softening, which was more conspicuous the longer the life of the animal was prolonged. The spinal cord above the middle of the dorsal region and the brain were normal in appearance, although scattering pellets were found here also.

It will be seen that Panum, in contra-distinction to Virchow, attributes the immediate cause of death in cases of rapidly fatal embolism to acute cerebral anæmia.

From a study of the literature on air-embolism, it is evident that the immediate cause of death has been assigned by different pathologists to one of the following conditions:—

1. Mechanical dilatation of the heart and paralysis of the organ in the diastole.
2. Acute cerebral ischæmia.
3. Asphyxia, resulting from mechanical obstruction to the passage of the blood through the pulmonary circulation.

As we shall see further on, death from air-embolism is not

always produced in the same manner; the mode of dying varies and is modified :—

1. By the amount of air admitted.
2. The time which has elapsed between the ingress of air and the fatal issue.
3. The location and distribution of the emboli.

II. *History of Air-Embolism.*

Surgeons and pathologists have for a long time been aware of the deleterious effects of free atmospheric air in the vascular system. The danger attending the forcible insufflation of air into the veins of animals was well known to many of the earlier physiologists. Among the first to study the effects of the introduction of air into veins may be mentioned Redi, Wepfer, Camerarius, de Heyde, Harder, Bohnius, Boerhaave, Lancisi, Morgagni, Valsalva, Bichât, and Nysten. As early as 1667 Redi killed animals by intravenous injections of air. He observed during his experiments that the pulse became intermittent, an occurrence which he attributed to the passage of a large air bubble through the heart. His followers who repeated the experiments soon discovered that after forcible insufflation of air into veins the air became diffused, inasmuch as at the post-mortem examinations they found it present in the right auricle, the coronary vessels, and in the shape of air bubbles, in the smaller vessels.

Merg made the observation that in opening the abdomen of a dog and puncturing the vena cava above the origin of the emulgents, as the vein became emptied of blood it filled with air which ascended with the blood current and entered the right side of the heart. Haller witnessed the same phenomenon in cold-blooded animals after wounding some of the large venous trunks. He has shown that it was from this source that the air was derived which Redi, Caldesi, and Morgagni had seen circulating in the vessels of the same animals. He claimed that air is never seen in vessels when the necessary precautions are exercised to prevent its introduction through a wounded vein.

Nysten found, by injecting air slowly into a vein, so as not to produce the death of the animal, that the coloring of the arterial blood was rendered imperfect. He satisfied himself that this change was not owing to the embarrassment of respiration. Insufflation of oxygen had no effect in preventing or correcting this change of color in the arterial blood. The literature on insufflation of air into veins is quite prolific and this subject cannot be justly passed over without an allusion to the following names:—

Blochmann. Aër in venis causa mortis. Dresden, 1843.
Bouillaud. De l'introduction de l'air dans les veines. Paris, 1838.
Gain. De aëris ingressione in venas. Berlin, 1865.
Maguin. Étude expérimentale sur l'introduction forcée et sur l'entrée spontanée de l'air dans les veines. Nancy, 1879.
Méric. Recherches sur l'introduction de l'air et des gaz qui le constituent dans le système veineux. Paris, 1866.
Valkenhoff. De aëris in venas ingressu ejusque effectu lethali. 1840.
Laborde. Effets de l'introduction de l'air dans la circulation artérielle. Compt. Rend. Soc. de Biolog. Paris, 1873.

These names are intimately associated with the experimental part of the history of air-embolism.

It was not long after the deleterious effects of free atmospheric air in the veins of animals had been studied experimentally before the same symptoms were observed in man by the accidental admission of air into wounded veins during operations, and in some of the first cases the presence of air in the veins and right side of the heart was demonstrated by post-mortem examination. Although a number of honest and reliable surgeons, prominent among them Velpeau and Fergusson,[1] have denied that a sufficient amount of air can be admitted through a wounded vein to produce sudden death, this assertion is no longer tenable in the face of such a large number of well-authenticated cases as have been recorded in surgical literature by equally conscientious and competent observers. Since the publication of the first well-authenticated case observed by Beauchène and described by

[1] Lettre sur l'introduction de l'air dans les veines de l'homme. Gaz. Méd., pp. 113–121. Paris, 1838.

Magendie the following authorities, placed in alphabetical order, have reported similar cases.

Assmus. Zur Casuistik des Lufteindringens in grössere Venenstämme. Med. Zeitung, xi. p. 104. Berlin, 1842.

Amussat. Introduction de l'air dans les veines. Bulletin Acad. de Méd., Paris, 1836, i. pp. 894, 899; 1837–38, ii. pp. 363, 461.

Barlow. An Attempt to Remove a Tumor on the Neck; Entrance of Air in Vein; Sudden Death. Med. Chir. Trans., xvi. pp. 28–35, 1830.

Chassaniol. Observation de l'entrée de l'air dans les veines pendant l'amputation du bras, dans son articulation scapulo-humérale. Union Méd., viii. p. 428. Paris, 1869.

Clémot. Lanc. Franc., tom. i. p. 357, 1830.

Coolidge. Case of Sudden Death from Entrance of Air into the Jugular Vein. New York Med. Gazette, i. p. 305, 1841–2. Also New York Med. Journal, vol. ix. pp. 199–201, 1847.

B. Cooper. Case of Alarming Syncope from the Admission of Air into a Vein, during Amputation of the Shoulder-Joint. London Lancet, i. pp. 448–451, 1843.

Cormack. Case of Death from the Entrance of Air by a Rigid Vein in the Neck, opened accidentally by a Seton-Needle. London Med. Journal, 1850.

Delaporte. Extirpation l'une tumeur située au cou; introduction de l'air dans le système vasculair. Bulletin Acad. de Méd., i. p. 132. Paris, 1836.

Delpech. Mém. des hôpitaux du midi, No. 16, p. 231, 1830.

Fischer, H. Ueber die Gefahren des Lufteintritts in die Venen während einer Operation. Volkmann's Sammlung klin. Vortraege. Chirurgie, No. 34.

Gunn. Syncope from Entrance of Air into the Facial Vein. New York Medical Journal, p. 356, 1852.

Heckford. Four Cases of Entry of Air into the Circulation. Medical Times and Gazette, i. p. 137. London, 1867.

Koestlin. Ein Fall von Luft im Herzen. Med. Correspondenzblatt d. württ. ærztl. Ver., xxviii. pp. 316–321. Stuttgart, 1857.

De Lavacherie. De l'opportunité d l'extraction des tumeurs du cou non susceptibles de resolution; reflexions sur l'introduction de l'air dans le cœur par des veines ouvertes accidentellement. Mém. Acad. Roy. de Méd. de Belge, ii. pp. 305–376. Bruxelles, 1849.

McPharlin. Death from Entrance of Air into the Veins in a Case of Compound Fracture. Hosp. Gazette, iii. p. 20. New York, 1878.

Massart. Etude nouvelle sur l'entrée de l'air dans les veines, dans les cas de plaie ou d'opération chirurgicale. Annales Soc. de Méd. d'Anvers, xv. pp. 5, 57, 113, 1854.

Mercier. Journal des connaiss, p. 108, September, 1836.

Meyer, F. Case of Injury of the Vena Jugularis Interna; Entrance of Air; Sudden Collapse; Recovery. Med. Archives, iii. pp. 408–410. St. Louis, 1869.

Miner. Tumor in the Neck; Admission of Air into the Vein; Death. Buffalo Med. and Surg. Journal, pp. 336–338, 1864.

Mirault. Thèse. Paris, 1832.

Mott. Entrance of Air into the Facial Vein. Medico-Chir. Trans., 1830.

Piachaud. Mort par introduction de l'air dans une veine pendant l'ablation d'une tumeur du sein avec ganglions dans l'aisselle. Echo Méd., p. 768. Neuchât, 1857.

Porter. On the Entrance of Air into the Veins as a Cause of Death. Journal American Med. Asso , iii. No. 20.

Rauch. Lufteintritt in einen verletzten grösseren Halsvenenast und seine Folgen. Oest. Med. Wochenschr., pp. 199–201. Wien, 1845.

Roux. Journ. Hebdom., ii. p. 64. 1833.

Schmid. Das Eindringen von Luft in eine Vene während einer Operation am Halse. Corresp. blatt d. württ. ärzt. Ver., xxi. p. 53. Stuttgart, 1851.

Schweickhart. Eindringen von Luft in die Venen; Tod durch Gehirnschlag. Mitth. des badischen ärzt. Ver., vi. pp. 69–71. Karlsruhe, 1852.

Smith, R. W. Abscess behind the Pharynx; Entrance of Air into Veins. Dublin Quarterly Journal Med. Sciences, xxv. p 497, 1844.

Tadlock. Entrance of Air into Divided Internal Jugular Vein; Ligation; Recovery. American Journal of Medical Sciences, p. 280, 1875.

Ulrich. Tod durch Eintritt von Luft in die Venen. Med. Zeitschrift des Vereins für Heilkunde, p. 132, November, 1834.

Warren, J. C. Two Cases of Accidents from Admission of Air into the Veins during Surgical Operations. American Journal of Medical Sciences, pp. 545–548, 1832.

Warren, J. M. Tumor connected with the Sartorius Muscle; Secondary Cancer of Breast; Operation; Entrance of Air into the Vein; Recovery. Surgical Observations, p. 529. Boston, 1867.

Wattmann. Prager Viertelj., ii. p. 191, 1844.

This list is, of course, not complete, nor does it represent all cases of accidental introduction of air during operations; but the names which are quoted ought to be accepted as sufficient guarantee by the most skeptical that the fear of this accident is not a myth but a reality substantiated by many a sad experience.

III. *Intravenous Production of Air.*

Spontaneous production of air within the bloodvessels of recently deceased persons has been repeatedly observed, and to it has been assigned one of the causes of sudden death. That the air thus produced is a direct product from the blood appears to be negatived by the fact that its occurrence has usually been traced in connection with sudden and exhaustive hemorrhages. It is, in fact, in persons who have died from hemorrhage, that

air has been found in greatest abundance in the veins. Lieutand[1] reports the case of a girl who died suddenly in a state of syncope, after having been repeatedly bled, and in whom the cerebral veins and choroid plexus were found impacted with air. M. Rerolle[2] has published several cases of the kind, where profuse hemorrhage had existed; in one of fatal epistaxis, the heart, arteries, and veins contained large quantities of air. Dr. Graves has noticed emphysema of the abdominal parietes in a sufferer from repeated attacks of epistaxis. M. Rerolle conjectures that, in such cases, air is absorbed by the radicles of the pulmonary veins—the air would have no claim to be considered adventitious.[3] It is, however, more logical to assume that, inasmuch as in almost all cases the supposed intravenous origin of air took place consequent upon severe losses of blood, hence likewise in connection with loss of continuity of the vascular system, that, owing to the sudden loss of intravascular pressure, the air may have been aspirated through the openings of some of the bleeding vessels. The quantity of air found in these instances has been so small that it has been impossible to make a chemical examination to determine its identity with atmospheric air. In cases where air was found in blood without loss of continuity of the vessels, it is not impossible that the supposed air was not atmospheric air, but a gaseous product liberated from the blood or generated in the tissues, producing a gas-embolism which interferes with the function of circulation in a similar manner as when the obstruction is caused by atmospheric air.

IV. *Effect of the Heart and Respiration on the Venous Circulation.*

As the state of the intravenous blood pressure constitutes the most important element both in the prevention and causation of aspiration of air into veins, this subject must be briefly alluded to in order to determine the conditions which act as exciting causes. For the most reliable and comprehensive information

[1] Hist. Anat., Med. Obs. 55. [2] Thèse de Paris, No. 129, 1832.
[3] Todd's Cyclopædia of An. and Physiol., vol. iv. part i. p. 145.

on this subject we are indebted to Jacobson.[1] The observations were made on sheep. To determine the effect of the heart's action upon the venous circulation he measured the blood-pressure in veins with the manometre. These measurements were made on veins in close proximity to the heart, as the lower portion of the jugular and subclavian, as all attempts to approach nearer the heart seriously impaired the normal physiological conditions of the respiratory and circulatory organs. This measurement gave the following results :—

In the left vena anonyma . . .	—0.1 mm. Hg.
" " right " jugularis . . .	+0.2 " "
" " right " subclavia . . .	—0.1 " "
" " left " jugularis . . .	—0.1 " "
" " left " subclavia . . .	—0.6 " "

The following are his observations on some of the more distal veins in the same animal :—

In the external facial	+3.0 mm. Hg.
" " internal "	+5.2 " "
" " brachial	+4.1 " "
" a branch of the same	+9.0 " "
" the crural vein	+11.4 " "

The experiments of Ludwig and Mogk, although made in a similar manner, led to more variable and inconstant results, at one time they found the blood-pressure in the crural vein 6.8 mm. Hg., while on another occasion under similar circumstances it measured in the same vein only 1.9 mm. Hg. Donders explains this want of uniformity to the respiratory movements of the chest, believing that the aspiratory movements of the chest affect the venous circulation more than the *vis a tergo* from the capillary system. Poisenille claimed that in his experiments the manometre was affected by the respiratory movements of the chest only when it was inserted into veins in close proximity to the heart, as in the lower portion of the jugular and the external iliac veins, while in more distant veins the column of

[1] Dr. Heinrich Jacobson. Ueber Blutbewegung in den Venen. Virchow's Archiv, xxxvi. p. 80.

mercury was not affected by the movements of the chest. Volkmann obtained the following measurements:—

In the facial vein of a goat	41	mm. Hg.
" " jugular vein of a goat . . .	18	" "
" " metatarsal vein of a calf . . .	27	" "
" " jugular vein of a calf . . .	21.5	" "
" a subcutaneous vein of the neck of a horse	44	" "
" the jugular vein of a horse . . .	21.5	" "

Magendie found the blood-pressure in the external jugular vein of a dog 18 mm. Hg., and in the crural vein 50 mm., although the measurements of the intravenous blood-pressure taken by different observers are at great variance, and although their figures are indicative of the opinions held by the different experimenters as to the effect of respiration upon the return of venous blood, yet they all agree in locating the minimum degree of intravenous pressure in the veins nearest the heart. The effect of respiration on the venous circulation was thoroughly investigated by Magendie. He introduced an elastic tube into the internal jugular vein, and observed that blood would escape only during expiration. The same experiment was made on the crural vein by directing the tube towards the heart, followed by the same result. The suction force exerted during inspiration was sufficient to counter-balance the auricular contractions. In making these experiments air was frequently drawn into the heart during forcible inspiration. Barry introduced through the jugular vein of a horse a bent tube of glass, one extremity being passed into the right cavity of the heart, or the vena cava, and the other into a vessel containing a colored fluid. He found that with each act of inspiration the liquid rose in the tube, demonstrating the effect of a notable suction force. He found that this suction force was increased by preventing the entrance of air into the chest by the trachea. He was of the opinion that this force from the chest was exerted not only in the large veins near the heart, but throughout the entire venous system.

Schweinburg[1] has studied the effect of respiration on the

[1] Die Bedeutung der Zwerchfellcontractionen fuer die respirat. Blutschwankungen. Du Bois-Reymond's Archiv, 1881, p. 475.

circulation by producing paralysis of the diaphragm by section of the phrenic nerves. He states that when diaphragmatic respiration has been artificially arrested, the difference of blood pressure observed during respiration ceases entirely or nearly so. From this he concludes that the action of the diaphragm causes to a certain extent these differences. Even after opening the abdominal cavity the difference in blood-pressure is very slight. As the principal cause in abolishing the effect of respiration upon the circulation he looks upon the compression of the abdominal vessels during inspiration, causing the increase of blood pressure during inspiration and its diminution during expiration by diminishing intra-abdominal compression. If the jugular vein in an animal is exposed, direct observations show conclusively that the direct influence of inspiration cannot be felt much beyond these vessels. The flaccidity of the walls of the veins will not permit the extended action of any suction force, but the flow of blood in the distant veins is accelerated by the intermittent emptying of the veins by the respiratory act. Barry and Donders ascribe to the aspiratory function of the chest, the principal motor in the return of the venous blood. Donders estimated the aspiratory force of the inspiratory movements of the chest at 7 mm. Clinical observation and experimental research have established the fact that the venous circulation is directly influenced by respiration within a certain area, and that aspiration of air in the majority of cases takes place in those veins thus affected, thus constituting the justly and much dreaded "danger-zone." Instead of speaking of the effect of respiration on veins as a cause of aspiration of air, some authors speak of the vein pulse, and limit the danger-zone to such veins which pulsate. Under certain circumstances the pulsations of the arteries are communicated directly to the veins through the capillaries. In such instances it is necessary that the arterioles are relaxed, as has been ascertained by Bernard in observing the circulation in glands during their physiological activity. If a vein be opened in a gland during its physiological activity, the blood retains partly its arterial hue and escapes in intermittent jets, as from a divided artery. According to recent

physiological investigations veins continue to pulsate independently of the arterial system and the cerebro-spinal centres. Luchsinger[1] examined the venous pulsation in the wings of bats. Contrary to Schiff's observations he found it independent of the central nervous system. Division of the brachial flexus and separation of all tissue connections between hand and body, with the exception of the vessels, did not arrest it. If artificial circulation was established in the organ after amputation rhythmic venous contractions would be seen even twenty hours after death. Intravenous pressure was found to be of great importance in these experiments; as soon as it was increased the vein began to pulsate. The seat of these rhythmic contractions Luchsinger placed in the walls of the vessels, or rather in their muscular structures. They are probably regulated by the central nervous system. Slight increase of warmth and electric tetanization accelerate the contractions. High temperature causes diastolic stasis. Nitrite of amyl increases the pulsating only to arrest it later. Schiff has since satisfied himself that these pulsations continue after division of the brachial plexus and ligature of the vessels, and even in the veins in detached pieces of the bat's wing.

Brunton[2] has made the same observations on man in regard to the effect of increased intravascular pressure in producing venous pulsations in the larger veins. He finds that pulsation of the jugular vein is sometimes confined to one side, the left one. In one of his cases the jugular on the left side was much more distended than the right jugular, the distension increasing whenever the vein was compressed just above the clavicle. Whenever this compression was repeated in the rhythm of the pulse, the increase and decrease of the blood in the vein assumed the character of pulsation, and for this reason the author has arrived at the conclusion that the venous pulsation in such instances is caused by compression of the vena anonyma by the aorta. All cases of unilateral jugular pulsation observed by

[1] Von den Venenherzen in der Flughaut der Fledermaäse. Pflueger's Archiv, 1881, vol. xxvi.

[2] On Pulsations in the Jugulars and other Veins. Medical Press and Circular, July 2, 1879.

Brunton occurred in anæmic women. In one of these the pulsation took place only while the patient was affected by some emotional excitement, in another only during expiration. In rabbits the author has repeatedly observed rhythmical contractions of the pulmonary veins, the vena cava inferior, and the portal vein, occurring immediately after the death of the animals. These pulsations were present either after complete cessation of the heart's action, and sometimes even before death, and, as the pulsations were more frequent than the heart's action, it was plain that they occurred independently from any contraction of that organ. In consequence of long-continued pressure on a vein the author has seen tonic contractions to take place, especially in smaller veins, and this may explain the cause of some of the irregularities of the circulation and subsequent transudation.

Riegel[1] has made vein pulsation a special subject of investigation, and as the result of his researches he has come to the following conclusions: 1. There exists in the normal condition a pulsation of the jugular vein. 2. This normal pulsation is always anadicrotic, *i. e.*, its wave rises in two distinct intervals. The anadicrotic wave corresponds, in contra-distinction to the pulsation of the carotids, to the disatole of the heart. The short catacrotic line or wave corresponds to the systole, the anacrotic to the diastole of the heart. Synchronous with the systole, the contents of the vein are emptied into the heart, while during the diastole stasis takes place in the veins.

King,[2] in his interesting essay "On the Safety-valve Function in the Right Ventricle of the Human Heart," demonstrates the existence of venous pulsations in the veins of the hand, the median veins of the forehead, and the external jugular which he observed after a full meal. The pulsations were made plainly visible by taking a delicate thread of sealing wax about two inches in length, one end of which was fixed across the vein with a little tallow so as to make a long and excessively light lever, capable of indicating a very slight movement in the vessel.

[1] Zur Kenntniss von dem Verhalten des Venensystems unter normalen und pathologischen Verhältnissen. Berl. Klin. Wochenschrift, 1881, No. 18.

[2] Guy's Hospital Reports, 1837, p. 108.

The movements of the lever produced by the vein pulse corresponded in frequency with the pulsations of the arteries in the same vicinity, but did not correspond in time as the venous pulse followed the arterial systole, showing conclusively that it was not due to the impulse of an adjacent artery. The pulsations could only be caused by the arterial wave being continued to the veins through the capillary vessels. In certain pathological conditions independently of valvular lesion of the heart he noted a marked increase in the venous pulsations in the dorsal veins of the hand and other vessels distant from the heart. The subject of the vein pulse affords an interesting topic in physiology, but in connection with this paper it is only mentioned in order to show that the intravenous tension is only slightly affected by it, and consequently it can exert no direct influence in causing aspiration of air into veins. The venous pulsations which directly influence the return of the venous blood to the right side of the heart occur synchronously with the movements of respiration and are observed only in the veins which are in close proximity to the heart, and in venous channels with firm unyielding walls. The introduction of air can only follow in wounds of vessels where the intravascular pressure is subjected to great variations either from normal anatomico-physiological conditions or the result of pathological alterations. All causes which prevent a prompt collapse of the walls of a wounded vein must be considered as predisposing causes, while all conditions which tend to produce a vacuum in the wounded vein act as determining causes. The location of the former corresponds to the point of injury, while the latter are always represented by the aspiratory action of the chest during inspiration.

V. *Aspiration of Air into the Superior Longitudinal Sinus.*

Nearly all of the older physiologists were of the opinion that aspiration of air into veins could only take place in vessels which were in close proximity to the heart and within reach of the venous pulse. Mery claimed that the effect of thoracic

aspiration on the venous circulation extends to the sinuses of the dura mater and the venous channels in the diploë of the cranial bones. Bernard was aware that air might enter the sinuses in case these structures were wounded, as this accident occurred a number of times in his experiments on animals where the superior longitudinal sinus was opened for other purposes. He believed that the air, after entering the sinus reaches the heart through the vertebral veins and the vena azygos. Death in such instances took place in eighteen minutes, while forty-five minutes were required if death resulted from hemorrhage alone.

Volkmann's case, reported in another part of this paper, demonstrates to a certainty that death may be caused by the entrance of air through a wound of the longitudinal sinus, and, although this is the only authenticated case on record, similar cases have undoubtedly occurred before, but the real cause of death was not recognized, and the fatal result was attributed to some other source. This subject of aspiration of air into the longitudinal sinus was made the object of experimental inquiry by Genzmer, one of Volkmann's assistants.[1]

The experiments were made on dogs, as this vessel in rabbits was found too small for the operation. Nine experiments were made. The animals were made partially insensible by morphine injections. The skull was exposed by an incision which was carried from the occipital bone to the forehead; with a small straight chisel, a section of bone about 6 cm. square was mapped out by cutting through the external table, anteriorly to the prominentia occipitalis externa, and which was completely detached with a hollow chisel. The dura mater having thus been freely exposed the posterior portion of the longitudinal sinus, which was about 2 mm. in width, was made accessible about its middle. Between two small hooks the sinus was made tense and divided transversely, carefully guarding against injury to the subarachnoidean space. In some of the experiments the

[1] Exstirpation eines faustgrossen Fungus durae matris, tödtlich verlaufen durch Lufteintritt in den geöffneten Sinus longitudinalis. Verh. d. deutschen Gesellschaft f. Chirurgie, vol. vi. p. 32.

wound was kept patent by making traction on its margins with the hooks, in others this precaution was unnecessary as the edges of the wound retracted sufficiently to keep it open. For several minutes after incision the bleeding continued profusely; the blood was quite red and escaped with some degree of force, the pulsations being plainly visible and synchronous with the heart's action. The stream was also perceptibly increased and diminished with the respiratory movements of the chest. After a few minutes had elapsed the hemorrhage became less profuse. In case the animal died, the heart and lungs were removed, after carefully tying the large vessels so as to prevent the escape of air from the heart. To secure accuracy in ascertaining the presence of air in the heart, this organ was opened under water when the rising bubbles would indicate its presence. In three cases, in two of them the animal breathed through a tracheal canula, the double rhythm in the blood column was lost soon after the sinus was opened, and the blood continued to flow until the animal died, which was usually the case after thirty-five, forty, and fifty-three minutes, the stream from the peripheral end of the sinus growing constantly less during this time. In all of these cases the central end of the sinus was completely filled with a thrombus, and no air was found in the heart. In two other cases the double rhythm continued until life was extinct, which was the case after twelve and nineteen minutes. After the first two or three minutes the bleeding diminished, and, by removing the blood from time to time with a sponge, it could be seen how air was aspirated during inspiration through the gaping wound. During forcible expiration, or on compressing the chest, air bubbles escaped with the blood from the wound, from the proximal end of the sinus. As the bleeding diminished air aspiration became more copious and more frequent. An examination of the cadavers of these animals revealed that the right side of the heart contained air and spumous blood. In the next two cases artificial dyspnœa was produced, in one instance by dividing both pneumogastric nerves, in the other by closing the tracheal canula through which the animal was breathing. In the first case air entered early and the animal died in sixteen

minutes; in the second case air entered freely during the forcible inspiratory efforts; the animal died in twenty-four minutes. In both of these cases air was found in the right side of the heart and in the subpleural vessels. In the last two experiments the animals were killed fifteen and sixty minutes after the sinus was opened, by puncturing the brain with a needle. In the first case a considerable amount of air was found in the right side of the heart, and in the second case the amount of air contained in the right side of the heart was less, the apparent difference being due to the presence of a thrombus in the central end of the sinus in the last case which prevented further ingress of air.

In recapitulation it may be stated that in six out of nine experiments air entered the longitudinal sinus, thus proving conclusively that wounds of this great reservoir of venous blood are not only dangerous from the loss of blood, thrombosis, and inflammation, but may also become the direct cause of sudden death by admitting air into the venous circulation.

VI. *Experiments.*

These experiments were made by the writer for the purpose of ascertaining more fully the conditions which determine the entrance of air into a wounded longitudinal sinus, and, at the same time, to obtain reliable information concerning the prophylactic measures as well as to determine the best methods of arresting hemorrhage in wounds of this vessel. All operations were made under antiseptic precautions; when not specified, no anæsthetic was used. The field of operation was cleanly shaved, and the surface thoroughly disinfected with a five per cent. solution of carbolic acid; during the operations, frequent use was made of the irrigator, using a warm two per cent. solution of the same antiseptic. When the animal survived the operation, the wound was closed with continued catgut sutures, dressed with iodoform, and a compress of salicylated cotton retained by a roller bandage. The operation consisted in making a longitudinal incision in the median line of the skull, reaching from the external occipital protuberance to near the upper extremity of

the frontal sinuses. The soft parts with the periosteum were separated and reflected on each side so as to lay bare the bone over a sufficiently large area for the ready use of the bone-cutting instruments. A medium-sized trephine was applied over the middle of the longitudinal sinus, and the button of the bone carefully removed so as to prevent injury of the underlying vessel. The enlargement of the circular aperture was effected with a hollow chisel and Luer's bone-forceps. The opening in the bone was made of an oval or oblong shape with the longest diameter parallel to the sinus, in order to bring into view a large extent of the vessel with a minimum destruction of the cranial vault.

Experiment No. 1.—Small Skye terrier, weight 12 pounds. Ether used as an anæsthetic. Longitudinal sinus laid bare to the extent of one and one half inches by an oval opening in the skull. Copious hemorrhage from a vein leading into sinus which was arrested after ligature. Two catgut ligatures were placed underneath the sinus about one-half inch apart, and the vessel cut transversely between them. The bleeding was very copious, the blood escaping in jets synchronous with the heart's impulse, the flow was also distinctly increased and diminished by the respiratory movements of the chest. During inspiration the stream was diminished while expiration was always attended by a decided increase in the force of the jet and the amount of bleeding. No air was seen to enter, although the hemorrhage had been very profuse and continuous for a considerable length of time. As it was intended by this experiment to prove that sudden obliteration of the longitudinal sinus is not incompatible with life, the distal ligature was tied with the effect of nearly but not completely arresting the hemorrhage, as some blood escaped from the proximal end of the vessel. It was now expected that air would be more prone to enter through the gaping wound in the sinus as the blood pressure from the distal end of the vessel had been arrested by the ligature, but as this accident did not take place after a few minutes the second ligature was tied, and the wound in the skin united with the continued catgut suture, and the antiseptic compress applied. The animal showed no other symptoms except great prostration from the sudden and profuse loss of blood. After an hour it rallied and apparently was in full posses-

sion of all its special senses, and was able to walk about as usual. The next day it manifested a ravenous appetite, and, during the whole time it was kept under observation, it showed no signs of illness or discomfort. The wound united by primary union, the skull showing the oblong bony defect at the site of the operation through which the pulsations of the brain could be distinctly seen and felt. Unfortunately the animal ran away after complete recovery had taken place and deprived me of the opportunity to study by post-mortem examination the local effect on the intracranial circulation by the operation. This experiment tends to prove that ligature of the longitudinal sinus can be performed without seriously compromising the functions of the brain, and that in certain well-defined instances this procedure might be resorted to in practice with a view of preventing or arresting hemorrhage from, and the entrance of air into, this vessel, in intentional or accidental wounds of the sinus.

Experiment No. 2.—Small tan cur, weight 10 pounds. Partial ether anæsthesia. Longitudinal sinus opened by two transverse incisions in close proximity; hemorrhage alarming, at first in jets, and, as the bleeding diminished, in a more continuous flow. At first the blood was bright red, but as respiration became impaired, it grew darker in color. Dilating forceps were introduced into the proximal wound, the hemorrhage continued, but no air entered as long as the animal was in a lying position, but as the respiration became more irregular and superficial artificial respiration was resorted to, and the head placed in an elevated position, whereupon the heart suddenly ceased to pulsate, and, upon applying the ear to the precordial region, a few irregular and very feeble contractions were heard, attended by a distinct churning sound, when the animal suddenly expired. Before death electricity was used with the effect of improving the respirations, but it had no effect whatever upon the action of the heart. Death took place about three-quarters of an hour after the sinus was opened. At the examination, immediately after death, all the tissues and organs were found in an exsanguinated condition. All the vessels leading to and from the heart were carefully tied, and the organ removed. On being placed in water it floated like a cork; the right auricle and ventricle were dilated, and on being opened under water bubbles of air and only a very slight amount of spumous blood escaped. The pulmonary

artery was also distended with air. The left ventricle was almost completely empty. In this instance the animal almost bled to death from the wounds in the longitudinal sinus, and yet no air entered, although the wound was kept patent with a pair of forceps. The entrance of air was caused by the elevation of the head and the forcible movements of the chest during the performance of artificial respiration. To judge from the amount of air found in the right side of the heart and its effects, the air must have entered quickly and in considerable quantity, distending at once the right side of the heart to such an extent as to paralyze the muscles of the heart in the diastole, after a few feeble attempts to force it from the right chambers. I believe, if the animal had been left in the lying position, and the head dependent, that death would have taken place from hemorrhage, as the blood which was draining through the sinus prevented the entrance of air, but as soon as the head was raised, the contents of the sinus by gravitation flowed towards the heart, and air entered with it to fill the vacuum which was being prepared by the diminished blood supply to the brain, and the acceleration of venous return, as well as the increased aspiration of the chest, which was brought about by the attempts at artificial respiration.

Experiment No. 3.—Horse, about 12 years old. Partial chloroform anæsthesia. Animal kept lying on the ground, head even with the body. Longitudinal sinus exposed for about two inches and incised longitudinally one inch Hemorrhage very profuse; blood at first bright red, gradually growing darker in color; double wave well marked. After about three quarts of blood had been lost, and the hemorrhage still continuing at the same rate, and not being readily controlled by the ordinary compression, it was decided to implant an aseptic sponge into the sinus. This was done, and the external wound united over it by the continuous suture. No air was seen to enter the wound, and auscultation over the heart revealed no abnormal sounds. During the operation of chiselling, the apices of the frontal sinuses were opened, which led to the fear that infection of the wound would subsequently take place from this source. This expectation was realized. The animal rallied soon after the operation, and appeared to be quite well for three days subsequently, grazing in the pasture with other horses. On the morning of the fourth day it was found dead. Examination of the cadaver showed

that the proximal end of the sinus was closed by a thrombus firmly adherent to the walls of the vessels and the implanted sponge, but about the distal end of the sponge, at a point which corresponded to the opening in the frontal sinuses, the brain and meninges showed all the appearances of acute septic inflammation. If infection had not taken place the aseptic sponge would have fulfilled all the purposes for which it was intended—arrest of hemorrhage and obliteration of the sinus. It seems to me that in cases of uncontrollable hemorrhage from accessible venous sinuses, the implantation of an aseptic sponge would prove a safe and efficient measure against hemorrhage, and would offer no obstacle against obtaining primary union and definitive closure of the vessel, as during the process of granulation the sponge disappears by absorption.

Experiment No. 4.—Horse, 14 years old, in good condition. This experiment was made for the purpose of confirming the suspicions already gained that the force of gravitation constitutes the most important factor in determining the admission of air into an open sinus of the dura mater; consequently no anæsthetic was given, but the animal was firmly held by a bit, and the operation was performed, without any difficulty, while the animal was in a standing position with the head elevated. With the trephine and chisel an oval opening, about two and a half inches in extent, was made over the longitudinal sinus. After all oozing had ceased, and the sinus being fully in view, its anterior wall was divided completely in a transverse direction. The edges of the wound immediately retracted, forming a diamond-shaped opening through which blood escaped in moderate force, but not nearly as copiously as on previous occasions when the animals were in the lying position. During the first inspiration after incision air entered with a loud gurgling or lapping sound, and on applying the ear over the apex of the heart a loud churning sound was heard synchronous with the movements of the heart. During expiration air-bubbles were seen to escape from the proximal end of the sinus. As soon as the head was depressed the hemorrhage greatly increased, but air never entered in this position; but as soon as the head was elevated, hemorrhage either ceased entirely or was at least greatly diminished, but air was sure to enter during inspiration. These manœuvres were repeated a number of times, and always with the same results. As the amount of air which was aspirated increased, the respirations

became more labored, and signs of cyanosis became apparent. An attempt was now made to close the wound in the sinus by sutures, and in this way arrest the hemorrhage. Three catgut sutures were passed through both edges of the wound, but on attempting to approximate its margins every one of them tore through the tissues before the parts were in apposition, proving conclusively that transverse wounds of the longitudinal sinus cannot be sutured, owing to the unyielding nature of the tissues. The external wound was completely closed by the continuous suture, and a firm graduated antiseptic compress controlled the bleeding. During the whole time of the operation, which lasted over an hour, some one of the bystanders listened to the heart's action, and the loud splashing or churning sounds were constantly heard. When the animal was released it commenced grazing in the pasture, and appeared as well as before the operation. The heart was examined at intervals of thirty minutes, and the abnormal sounds grew more feeble, and after an hour had entirely disappeared. The sound produced by the entering air, I have described as lapping, resembling very much the sound produced by the lapping of a dog or cat; the best possible word for this sound is the German expression "schluerfend." When air enters through a wound of the longitudinal sinus this sound is characteristic, and is always the same, and, in case the animal operated upon is a horse, it is sufficiently loud to be heard at some distance. Experiments have shown that horses are most tolerant to the presence of air in veins, on account of the unusual development of the right ventricle, which has sufficient power to force the air through the pulmonary circulation, and this experiment would certainly tend to corroborate this observation, as air in large quantities was aspirated at least a dozen times during the operation, and that most of it entered the right side of the heart, and was not returned is evident from the persistence of the sounds, due to the presence of air for a period of two hours, and yet, aside from a certain degree of embarrassment of respiration, the animal suffered no inconvenience. The wound healed by primary union. The defect in the skull remained permanent. The animal was killed about four weeks afterwards. Post-mortem appearances: The trephine opening filled with cicatricial tissue. Proximal end of sinus, just behind trephine opening, contains one large granulation thrombus. Cicatricial tissue filling almost the entire lumen of the sinus. Anteriorly the sinus is somewhat contracted and smooth; no

thrombus here or evidences of proliferation. The circulation is apparently restored by the formation of a new channel or dilatation of a pre-existing one; this new sinus is located to the left of the median line. The lateral sinuses are very much enlarged.

Experiment No. 5.—Young yellow dog, weight about 15 pounds. Partial ether anæsthesia. Longitudinal sinus exposed and transversely incised at two points in close proximity. Hemorrhage profuse, was allowed to continue for over half an hour in order to estimate the length of time which would be necessary for death to occur from this cause uncomplicated by admission of air. When the animal appeared moribund both ends of the sinus were ligated. The heart's action was very feeble and irregular while respiration was entirely suspended. Artificial respiration was kept up until the heart's action ceased. Death occurred in thirty-five minutes. At the examination after death no air was found in the vessels or heart, and death was plainly attributed in the absence of any other cause solely to the loss of blood.

Experiment No. 6.—Newfoundland dog, weight 50 pounds. Partial ether narcosis. During the removal of bone over the sinus severe hemorrhage was encountered from the large venous channels in the diploë. The great irregularity of the external surface of the skull led to a mistake, as the frontal sinuses were again opened. The longitudinal sinus was laid open by an incision half an inch in length in a parallel direction to the vessel. Hemorrhage very profuse for half an hour, checked at times by compression, when it finally diminished and the wound was closed. After the operation the animal walked with a staggering gait, and would run against objects indiscriminately, showing that sight was greatly impaired, inasmuch as the animal had fully recovered from the effects of the ether. No air was seen or heard to enter the sinus. Heart sounds feeble, but otherwise normal. Death in this case took place a week after the operation from lepto-meningitis. The source of infection undoubtedly was again traceable to injury of the frontal sinus, as the earliest evidences of the disease were found nearest the opening in this structure.

Experiment No. 7.—Old decrepit horse. Operation was performed while the animal was in the erect position. On removing

disk with trephine a longitudinal wound one-half inch in length was found in the anterior wall of the sinus through which bright red blood escaped. Double pulsation well marked. Almost immediately after the removal of the disk of bone, and before more than an ounce of blood had escaped, air entered with a loud and distinct lapping sound, audible to all who were present. On applying the ear over the heart the same loud churning sounds were heard. As the head was lowered the flow of blood became more forcible and copious, but no air entered; as soon, however, as the head was elevated, bleeding diminished, and air entered during almost every inspiration. Respiration became labored, and after air had entered four or five different times in succession the animal fell to the ground. In this position no further entrance of air occurred, but the hemorrhage continued copiously, the blood flowing in a continuous stream with a well-marked double jet synchronous with the action of the heart and the respiratory movements of the chest. The opening in the skull was enlarged to two inches in length and one and one-half inches in width, so as to expose the sinus freely. A number of catgut sutures were now introduced through both lips of the wound, and on attempting to tie them great difficulty was experienced in approximating its margins, which could be brought nearly but not completely in contact without the sutures tearing through. The tying of all the sutures resulted in diminishing but not arresting the bleeding, showing conclusively that in longitudinal wounds of the sinus suturing is an imperfect and unreliable measure in arresting hemorrhage, to say nothing of the difficulty which is experienced in passing the sutures at such great depths and in the limited space furnished by the artificial opening in the skull. The wound was not closed but tamponed with iodoform cotton in order to observe from time to time the processes which nature would initiate in the restoration of the wounded sinus. Half an hour after the first entrance of air the churning sounds in the heart had much diminished, and almost completely disappeared after the lapse of one hour. The animal recovered completely from the immediate effects of the air-embolism, the respiration having again become normal in frequency and character. After two hours the tampon was removed, but bleeding again occurred, and it was replaced. The animal died twenty-four hours after the operation, probably from the combined effect of loss of blood, hemorrhage into the subdural spaces, air-

embolism, and senile marasmus. At the examination of the cadaver a subdural clot was found on the right side of the brain which weighed about half an ounce, on the left side of the sinus a second but smaller subdural clot was found. Trephine opening filled by a coagulum. One of the sutures had lost its hold by tearing through the tissues of one margin of the wound. Within the sinus a small fragile clot was found in the lateral wall of the sinus which served as the source of hemorrhage into the subdural space. This experiment illustrates well the danger of plugging the opening in the skull for the purpose of arresting hemorrhage in case the lateral walls of the sinus are injured, as it will almost necessarily lead to subdural hemorrhage and expose the patient to all the disastrous consequences incident to this occurrence. In this instance sponge implantation would not only have more successfully guarded against external bleeding, but would also have served as a sure prophylactic against extravasation into the subdural space. It also teaches that suturing in cases of wounds of the longitudinal sinus with limited defects in the bony walls of the cranium is impracticable, unreliable, and unsafe, and should never be resorted to unless the dura mater is so extensively exposed or separated as to permit, by making gentle traction, perfect and complete approximation of the margins of the wound.

VII. *Practical Suggestions.*

In order to study the conditions which favor the aspiration of air into a wounded sinus of the brain it is necessary to call attention to some of the peculiarities of the intracranial circulation. Mosso,[1] who has made this a special subject of investigation, asserts that the intravascular presence in the veins within the cranium is higher than in the veins of any other part of the body. Actual measurements have shown that the blood-pressure in the longitudinal sinus is equal to 100–110 mm. Hg. The probable cause of this phenomenon is that the force of distension of the arteries within the closed and unyielding cranial cavity is added to the *vis a tergo*. The intracranial veins show distinct pulsations which are dependent upon the pulsations of the arteries, and their movements are so plain that they can be

[1] Ueber den Kreislauf des Blutes im menschlichen Gehirn, 1881.

graphically demonstrated ; every diastolic movement in the artery corresponds to a venous pulse. During the pulsations of the brain, Mosso claims with Donders and Berlin, that the cerebro-spinal fluid does not escape into the spinal canal. In a case of spina bifida he has been able to trace respiratory but no circulatory movements. When the tumor was compressed only a very slight increase in the volume of the brain could be detected at the fontanelle, even if nearly the whole contents of the tumor were pressed into the spinal canal. G. Burkhart[1] has published the results of his observations on the movements of the brain which he made on four patients who had suffered partial loss of the cranial vault. The tracings obtained represented three forms of movements—pulsatile, respiratory, and vascular. The cerebral pulsation has the form of a tricrotic or tetracrotic pulse, the phases following one another in about the same time as those of the carotid pulse. His observations lead him to the conclusion that the brain presents the same movements within the intact skull as in the infant or when a defect in the skull exists, the result of traumatism. The brain expansion is synchronous with the dilatation of the vessels and takes place in the direction of the vascular ramifications. The resistance is in the inverse proportion to this expansion. In the closed skull the excess of pressure in the arteries aids materially the propulsion of the blood through the veins, and also that of the serolymphatic fluid. In the open skull the curve rises during expiration and falls during inspiration. All actions which increase the respiratory movements increase the height of the curve. A secondary elevation follows labored inspiratory movements, but the pulse waves are never completely effaced. The vascular curves occur independently of respiration or pulsation. The height of the curves bears no constant relation to their length. They are notably influenced by psychical influences. They are produced by movements of the vessels by means of the vasomotor nerves, and can be made very conspicuous by irritation of the cervical sympathetic. Bergmann,[2] in his remarks on the

[1] London Lancet, Oct. 15, 1881.

[2] Verhandlungen d. deutschen Gesellschaft f. Chirurgie, vol. x. p. 14.

movements of the brain (in opposition to Mosso) at the meeting of the Congress of German Surgeons in 1881, insists that the cerebro-spinal fluid acts as a regulator in maintaining the equilibrium between the arterial and venous circulation within the cranium. The pulsations of the sinuses of the dura mater were discovered and studied under his supervision at Dorpat as early as 1873. He argues that these pulsations are very slight, and on that account insufficient to counterbalance the arterial pulsations. He explains the pulsations of the sinuses in the same way as Donders and Jacobi have accounted for the pulsations in the veins of the papilla of the optic nerve. The pulsations are the result of increased tension in the cerebro-spinal fluid during the arterial systole and the consecutive diminution of intracranial pressure during the arterial diastole. The cranium being a closed cavity with unyielding walls, it is not difficult to understand that in case one of the sinuses is opened by a wound which communicates with the atmospheric air, the sudden loss of blood will have a tendency to create a vacuum which is filled by the admission of air which reaches the left side of the heart with the venous blood. All circumstances which diminish intravascular and intracranial pressure must of necessity favor the occurrence of aspiration of air into a wounded sinus. It is evident that aspiration of air into an open wound of the longitudinal or any other sinus of the dura mater is favored by the following conditions: 1. The force of gravitation. 2. The inspiratory movements of the chest. 3. The condition of arterial circulation. In considering the prophylactic treatment against the admission of air during operations which involve any of the cerebral sinuses, it is of the greatest importance to keep the head at a level with the heart to insure regular respiration and to guard against undue or forcible inspiration, and, finally, to maintain the normal activity of the ventricular contractions. The direct preventive measures consist in:—

1. Continuous irrigation of the field of operation. 2. Prophylactic ligation of the sinus.

In resorting to constant irrigation the fluid used should be an aseptic solution at the temperature of the body, which, if it

should enter the venous circulation to fill an empty space, would do no harm either as a toxic agent or by causing coagulation of the blood. A solution of salicylic acid in distilled water, or borated water would be best adapted for this purpose. In extirpating tumors of the dura mater in the region of the longitudinal sinus, where wounding of this structure becomes a necessity, it would not only be prudent but good practice to ligate the sinus on each side before attempting the removal of the tumor, as this precaution would surely and effectually prevent the two most dangerous and alarming accidents—hemorrhage and aspiration of air. This plan was followed by Kuester in removing a sarcoma of the dura mater in 1881.[1] Experiments on animals and the cadaver have convinced me that this operation can be performed with comparative ease, if the defect in the skull is sufficient in extent to permit the necessary manipulations, and, in the event this operation be done for the purpose of facilitating the removal of tumors of the dura mater, this precaution should never be neglected. With a tenaculum the dura mater is seized and drawn forward at the outer border of the longitudinal sinus, and a small incision parallel with the border of the sinus should be made with a tenotome, the incision being only sufficiently deep to divide the dura mater. After making such an incision on each side of the sinus directly opposite each other, the sinus should be grasped with a sharp-toothed spring forceps and drawn forward, when a small curved sharp-pointed aneurism needle is passed into one of the openings, and, after penetrating the falx cerebri underneath the vessel, is brought out through the opening on the opposite side. When both of the ligatures are in place the peripheral ligature is tied first, and after emptying the intervening part of the vessel of its contents the proximal ligature is also tied. If both of the ligatures have been properly applied the intervening portion of the sinus can be opened or excised with the tumor without risk of hemorrhage or the introduction of air, at the same time it will greatly facilitate thoroughness in the removal of diseased tissue. I am firmly

[1] Berl. Klin. Wochenschrift, p. 673, 1881.

convinced that the preliminary ligation of the longitudinal sinus will become an established procedure in all cases where tumors of the dura mater are so situated that this removal implicates this structure and that it will render possible the removal of tumors which without it would place in great and immediate jeopardy the life of the patient by hemorrhage or the admission of air. The process of cicatrization in the sinus is the same as in veins, and is accomplished in the same brief period of time. In accidental wounds of the sinus ligation should be resorted to whenever the original defect in the skull is sufficient to permit the necessary manipulations or when simpler measures have failed to accomplish the same object. Implantation of an aseptic sponge into a wounded sinus should be resorted to in all cases of wounds of the lateral walls of the sinus, in cases of accidental wounds where ligation is impossible and where other measures have failed to arrest the hemorrhage. The sponge should be large enough to make gentle pressure upon the inner surfaces of the sinus, and yet sufficiently firm to arrest the circulation in the vessel so as to prevent the escape of blood into the subdural space. The hæmostatic action of the aseptic tampon is made more efficient by adding external compression, applied in the form of a graduated aseptic tampon. If the wound remains aseptic the sponge forms a nucleus for the thrombus and is infiltrated by connective-tissue cells from the intima and adjacent tissues, and is gradually removed by absorption as the definitive obliteration of the vessel proceeds. Small wounds of the sinus can be readily and safely closed with the lateral ligature applied in the same manner as in similar wounds of the veins.

In recapitulation we are warranted in stating the following conclusions :—

1. Elevation of the head is the direct and most essential cause in the production of air-embolism through a wound of the superior longitudinal sinus.

2. Suturing of a wound of the superior longitudinal sinus as a hæmostatic procedure is unreliable, and in most instances anatomically impossible.

3. Prophylactic ligation of the superior longitudinal sinus

should be resorted to in all cases where this vessel is involved in extirpating tumors of the dura mater.

4. Implantation of an aseptic sponge into a wounded longitudinal sinus will arrest hemorrhage without interfering with the definitive obliteration of the vessel, and deserves a trial in cases where the lateral walls of the sinus have suffered injury and where ligation is impracticable.

VIII. *Immediate Cause of Death after Intravenous Insufflation of Air.*

Various theories have been advanced to explain the injurious effect of the presence of air in the circulation. Bichât[1] attributed death resulting from intravenous injection of air to cerebral anæmia produced by the presence of air in the cerebral vessels, asserting at the same time that a very small quantity would suffice to produce this effect. As the first argument in favor of this view, he claims that the heart continues to beat for some time after the cessation of animal life. Secondly, air injected through one of the carotids produces death in the same way as when introduced into the veins. Thirdly, the cases reported by Morgagni, where death was attributed to the presence of air which was found in the cerebral vessels at the post-mortem examination, and which was supposed to have developed there spontaneously. Fourthly, all examinations after death revealed the presence of frothy blood, mixed with air-bubbles in both ventricles. Fifthly, air injected into one of the divisions of the portal vein produces no ill-effects until it reaches the general circulation. Sixthly, the almost instantaneous death observed in some instances is due to the acceleration of the heart's action, and consequently the rapid conveyance of air into the cerebral vessels. Seventhly, the existence of convulsions which he ascribes to the irritating quality of the air on the brain. He summarizes as follows: "We shall conclude that in the accidental mixture of air with the blood of the venous system, it is

[1] Physiological Researches on Life and Death, p. 186.

the brain which dies the first, and that the death of the heart is the consequence of the death of the brain."

Magendie, in commenting on Bichât's views concerning the manner of death from intravenous injection of air, remarks: "This is not correct, and death takes place, on the contrary, by the cessation of the motions of the heart. The right ventricle is filled with air, and this air, dilated by heat, so distends it that it can no longer contract." Magendie also claimed that small quantities of air in veins will not result in death, and that life is in jeopardy only when the air is injected suddenly and in considerable quantity. He relates the details of an experiment which he made on a horse, where he injected in rapid succession into the veins of the animal forty syringefuls of air, and three syringefuls into the carotid artery. The capacity of the syringe was seventeen centilitres. The animal died three minutes after the last injection. At the examination of the body he found air in the azygos vein and in the thoracic duct which contained much lymph, as well as in the lymphatic vessels of the internal surface of the lungs. The heart was enormously distended with air mixed with a small quantity of blood. Morgagni, Brunner, Sprœgel, and Nysten referred the cause of death to the same source—over-distension and paralysis of the heart. When death is not produced by the mechanical effect of the air on the heart, then the consecutive symptoms were referred by Nysten to an obstruction of the lungs produced by the accumulation of air in the ultimate divisions of the pulmonary artery. He observed that the embarrassment in respiration often appears as late as twelve hours after the introduction of the air, and becomes greater and greater; the bronchi are filled with a viscid fluid, and the animal usually dies on the third or fourth day. In such instances no air was found in the heart or the vessels, but the lungs, instead of being pink-colored, were grayish, tinged with brown, and loaded with frothy blood and mucus. The same views were entertained by Boerhave, Kettler, and Beck. In such cases death results from asphyxia from the mechanical obstruction to the passage of the venous blood through the pulmonary circulation. Mery accepts the views of Mercier

published in 1839, who attributed death to this, that the blood mixed with air becomes frothy, enters the pulmonary capillaries and obstructs them. Physicists are aware that a capillary tube which readily admits the passage of air or water offers a great resistance to a mixture of air and water; this mixture causes a series of bubbles, separated by minute septa of liquid. Poisenille has shown by a series of experiments that such in fact is the cause of death whenever air mixed with blood has obstructed the pulmonary capillaries. The right heart remains over-distended and cannot be emptied.

Blundel[1] studied the effects of the intravenous injection of air in his experiments on transfusion of blood. He injected five drachms of atmospheric air into the femoral vein of a small dog in quantities of a drachm each at a time, without any serious effects. The symptoms observed were sighing respiration, irregular pulse, muscular tremors, and vomiting, all of which, however, subsided after a brief space of time, and the animal recovered completely in three days. A second experiment was made on the same animal by blowing three drachms of pulmonary air into the femoral vein without even producing much *temporary* inconvenience. He concluded that it seemed indisputable that small quantities of air may be introduced into the circulation without destroying life. Dr. Haighton made the same experiments with intravenous injection of air and with like results. A series of experiments were made by Panum.[2] The subject of the first experiment was a small dog in which five cubic centimetres of air were injected into the lower portion of the jugular vein. No symptoms followed immediately after the operation. Four days subsequently the weight of the animal was reduced from 3540 to 3030 grammes. The tissues around the point of puncture were inflamed. On the fifth day the animal was killed. The skin of the animal was dotted with spots of ecchymosis resembling the extravasations as they occur in the disease known as morbus macu-

[1] Medico-Chir. Trans., vol. ix. p. 65.

[2] Experimentelle Beiträge zur Lehre von der Embolie. Virchow's Archiv, vol. v. p. 499.

losus Werlhofii. In the vicinity of the wound, the tissues were emphysematous, emitting an offensive odor. Among other post-mortem appearances the lungs presented several gray superficial stripes and spots, 1–2 lines in diameter. In the middle of several of these patches were found empty spaces which Panum regarded as encysted air-bubbles. In addition small isolated nodules were also found which contained in their interior small bubbles of air. These nodules contained also numerous nuclei and fat globules. In the second experiment ten cubic centimetres of air were injected into the lower portion of the jugular vein of a small one-year old dog. During the first three days nothing was observed with the exception of rapid emaciation. On the fourth day the animal began to lose the hair not only in the vicinity of the wound, as in the first case, but over the entire surface of the body. Later inflammation and ulceration attacked the point of operation. On the eleventh day the animal was killed. The lungs again presented subpleural nodules, the size of a pin's head to that of a grain of sand, in which could be found large cells filled with fat molecules, granular cells, and fat globules in large number. Air could not be found in any of them, and their relation to the capillaries of the pulmonary artery could not be determined. In the third experiment 30 cubic centimetres of air were injected into the jugular vein without producing any alarming symptoms, except rapid and deep respiration. The next day no change was observed, but on the following day the animal was found dead. A careful examination revealed punctiform extravasations in different portions of the brain, stomach, and liver. The wound showed a healthy appearance. The lungs contained many very small yellowish-white nodules and points of extravasation one to two lines in diameter. The hemorrhagic infarcts contained each a minute cavity filled with air. The nodules also contained air. From these experiments it appears that the air-emboli which passed through the pulmonary capillaries produced local disturbances in the minute vessels in the skin and gastro-intestinal canal, while the air-emboli in the ultimate branches of the pulmonary artery gave rise to circumscribed foci of inflammation.

Picard[1] found after insufflation of air into the portal vein intense hyperæmia in the distal portion and radicles of the vessel, the same as after ligation. At the commencement of the insufflation the blood pressure in the femoral artery and one of the rectal veins was about the same, but after a while the pressure in both was simultaneously diminished, but the positive pressure lasted longer in the vein than in the artery. The action of the heart was increased, the respirations became slower, and the temperature in the rectum was gradually reduced. As an interesting physiological fact it is mentioned that after insufflation of air into the portal vein no sugar could be found in the liver, and the fibrin in the portal blood was diminished.

The experiments of Magendie, Bouillaud, and later those of Couty,[2] prove conclusively that in cases of entrance of air into a peripheral vein the air collects almost exclusively in the right side of the heart; only a minute quantity entering the left side through the pulmonary capillaries. Referring to this question Flint says: "The production of death from air in the veins is purely mechanical. The air, finding its way to the right ventricle, is mixed with the blood in the form of minute bubbles and passed into the pulmonary artery. Once in this vessel, it is impossible for it to pass through the capillaries of the lungs, and death by suffocation is the inevitable result if the quantity of air be large. It is because no blood can pass through the lungs that the left cavities of the heart are usually found empty."[3] If the quantity of air introduced is small, or the entrance repeated in small quantities, the air collects in the capillaries of the pulmonary artery as air-emboli, obstructing the circulation in the impacted vessels, but a sufficient number of vessels remain pervious to maintain the circulation, and life is prolonged until the equilibrium of the circulation is restored by the absorption of the adventitious air. No traces of air are found in the arteries, only a small quantity passing into the venæ cavæ through the tricuspid valve, which has been rendered insufficient by the

[1] Sur les injections d'air dans la veine porte. Gaz. Méd. de Paris, No. 6, 1873.

[2] Étude experimentale sur l'entrée de l'air dans les veines, Paris, 1875.

[3] Physiology of Man, Blood, Circulation, Respiration, p. 323, 1866.

presence of air. When a considerable amount of air has entered the right ventricle of the heart mixed with the blood, the air is separated from it, and, its specific gravity being less, it rises to the highest point of the chamber in contact with the anterior walls; besides it expands and by this over-distension it impairs the muscular contractility which even in a normal condition is insufficient to empty the cavity completely. The impediment to the circulation being the presence of the large air-embolus in the right ventricle, which on account of the higher temperature increases in volume, sudden death will take place in the diastole by an arrest of the heart's action from paralysis by over-distension. If the animal escapes instantaneous death from this cause, the heart is inadequate to force the blood from the right ventricle through the pulmonary circulation, as its efforts are expended in compressing the air; only a minute quantity of blood being forced into the lungs. In proportion as the amount of air and blood in the right ventricle increases, the right side of the heart is expanded and the volume of blood in the lungs and the left heart is diminished. At last the distended walls of the heart prevent perfect closure of the tricuspid valve, giving rise to venous pulsation, a constant symptom in all cases of air-embolism which prove rapidly fatal. On the advent of this complication the intra-arterial pressure in the pulmonary and peripheral arteries diminishes, which further enfeebles the pulmonary circulation, at the same time it produces acute anæmia of the brain, and death results from anæmia of the brain or asphyxia. Such is the mechanism of death in cases of entrance of air into the venous circulation, and at the same time it offers an explanation why in these cases the air is found mostly in the right side of the heart and the large venous trunks. These facts also corroborate the observations of experimenters that certain animals succumb more readily to the presence of air in veins than others. The tolerance of intravenous air is most marked in animals with well-developed respiratory organs and a proportionately powerful right ventricle. In horses, for example, the volume of the right cavities is smaller and the muscular structure more powerful, circumstances which

explain the fact that entrance of air into the veins of these animals does not easily kill them. The circulatory apparatus of the dog offers the least resistance to intravenous air. Rey blew air into the jugular veins of horses after venesection without producing death. Small amounts of air produced no result whatever, and a volume of air equivalent to two expirations proved fatal only in debilitated animals. Some animals remained well after leaving a canula in the jugular vein for several hours. Death was sure to follow if after the insufflation of air the vein was ligated. Animals who have previously lost large quantities of blood readily succumb to intravenous admission of air.

Laborde and Muron, who studied the effects of intravenous and intra-arterial insufflation of air, placed great stress on the manner in which the injections are made in determining the gravity of the symptoms. They observed that in case the insufflations into veins are made slowly and repeated at intervals large quantities were tolerated without exciting serious symptoms. If, on the other hand, they were made suddenly and with force, and the quantity of air was considerable, death resulted almost instantaneously from arrest of the heart's action in the diastole, an occurrence which was attributed by these authors to over-distension of the right side of the heart.

In repetition, it may be stated, that the immediate cause of death after intravenous injection of air has been referred by different experimenters to:—

1. Mechanical over-distension of the right ventricle of the heart and paralysis of this organ during the diastole.
2. Acute cerebral ischæmia.
3. Asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery.

IX. *Intra-Arterial Insufflation of Air.*

As the accidental admission of air never takes place in wounds of the arteries on account of the high degree of intravascular pressure and the absence of any aspiratory force it is not sur-

prising that the effect of the artificial introduction of air into these vessels has been made less frequently an object of experimental research than intravenous insufflation of air. The subject is devoid of any practical value, and the interest attached to it is of a purely scientific nature. As we have already seen, Bichât entertained the idea that atmospheric air acts as a direct irritant to the substance of the brain when brought in contact with that organ through the medium of the cerebral vessels, and death from insufflation of air, whether into veins or arteries, was invariably attributed by him to cerebral anæmia. Panum,[1] in his researches on embolism, made arterial insufflation of air also a subject of experimentation. A rapidly fatal termination followed the injection of 4 c. centimetres of air into the lower portion of the carotid artery of a medium-sized dog. The infundation was superseded immediately by severe general convulsions, alternated by violent attacks of rage. The animal foamed at the mouth, and had involuntary discharges from the bladder and bowels, the eyes became prominent, and the pupils, at first dilated, contracted, and remained so permanently. The animal soon became quiet and motionless. Respiration and heart's action slow. The right anterior and both posterior extremities were extended and rigid, while the left front leg remained relaxed. Conjunctiva insensible, while touching the cornea produced contractions of the eyelids. For two hours the animal remained in a condition resembling anæsthesia without changing its position, with slow pulse and respiration, when life ceased without a tremor or convulsion. The post-mortem examination showed numerous punctiform ecchymoses in the gastro-intestinal mucous membrane, the liver, diaphragm, and abdominal muscles. The superficial vessels of the brain, more particularly the veins, were extremely hyperæmic; the jugular veins were distended to their utmost with blood. The small arteries contained many air-bubbles, so that the smallest vessels presented varicose dilatations which resembled a string of pearls. The large vessels at the base of the brain also contained air, and

[1] Op. cit.

numerous red spots were disseminated throughout the white substance of the brain.

Laborde and Muron[1] witnessed after the introduction of 20–60 c. cm. of air into the carotid artery of dogs, when injected in a peripheral direction, that death was produced rapidly and was always preceded by tetanic convulsions and labored respiration, while if the quantity of air injected was smaller, and especially if the air was thrown in in divided doses, the animal often survived the experiment for twenty-four hours. In the latter class of cases the operation was followed by tetanus, vomiting, paralysis, and coma. The autopsy revealed softening of the brain, and capillary hemorrhages, especially in the middle portion of the brain, the medulla oblongata, and in the posterior lobes of the cerebrum. The intra-arterial insufflations of air in all these experiments were immediately followed by grave cerebral symptoms which can only be interpreted by the constant post-mortem appearances, air-embolism of the cerebral vessels, and extreme ischæmia of the brain. The presence of air in the arteries, on the left side of the heart, is followed by an entirely different series of phenomena than in the veins on the right side of the heart. The acute ischæmia of the brain thus induced is invariably manifested by tetanic rigidity of the voluntary muscles and almost complete suspension of the respiratory movements of the chest. The contractions of the left ventricle are so powerful as to overcome these additional impediments and to completely evacuate the chamber. The air is expelled as soon as it enters and is distributed throughout the whole arterial system. It must be readily conceived that the air when it has reached the aorta will rise into the carotid arteries and thence into the cerebral vessels, distending them to their utmost capacity. In such instances death results from sudden cerebral anæmia before the air can gain entrance into the venous system through the capillaries. That the presence of air is detrimental to the nervous system has been established by experiments on animals. Bohnius attributed to the air when introduced into the vascular

[1] Virchow u. Hirsch's Jahresbericht, vol i. p. 268, 1873.

system poisonous properties, and this opinion is entertained by Neudörfer even at the present time. Copeland believed that the oxygen of the adventitious air combines with the carbonic oxide in the venous blood producing carbonic acid. These and similar theories do not explain the phenomena observed after insufflation of air. The symptoms during life, and post-mortem appearances point directly towards physical obstruction in the bloodvessels by air-emboli which suspends the function of one of the vital organs, and consequently must be regarded as the immediate or direct cause of death. Insufflation of air into the carotid artery towards the brain is almost immediately fatal. If a moderate quantity of air is forced through the arterial system into any other organ except the brain, except by forming a temporary obstruction in the circulation, it does no particular harm and is in a short time removed by absorption. This fact may have induced Flint to make the following remarks concerning this subject:[1] "Air injected into the arteries produces no such serious effects as air in the veins. It is arrested in the capillaries of certain parts, and in the course of time is absorbed without having produced any injury."

The danger arising from the introduction of air into the arteries or left side of the heart arises from obstruction to the circulation through the cerebral vessels by air emboli and the cessation of the functions of the brain consequent upon an almost complete ischæmia of this organ. The mechanical distension of the left side of the heart by the accumulated air is overbalanced by the powerful contractions of the left ventricle, which are sufficient to empty the chamber almost completely, and to force the air into the ultimate distributions of the arteries causing acute anæmia in distant organs, but more particularly in the brain. In these instances the air is forced directly into the cerebral vessels either by the injecting force or the powerful contractions of the left ventricle, and if death takes place it follows as the direct result of acute cerebral ischæmia.

[1] Op. cit., p. 324.

X. *Clinical Study of Air-Embolism.*

All physiologists agree that regular and deep inspirations produce a powerful aspiration in the large veins near the base of the heart, more particularly in the jugular and subclavian veins. This fact is so well known that the base of the neck and the clavicular regions are frequently referred to as the "danger zone." During the inspiratory act the chest expands, and the flow of venous blood is accelerated towards the cardiac or proximal side. A diversity of opinion still exists among authors in regard to the distance to which this force directly affects the venous circulation. Experiment and clinical observation have shown that the danger of entrance of air into wounded veins is increased as the wound approaches the heart. Berard studied the anatomical conditions of the vessels where entrance of air has most frequently taken place, and he came to the conclusion that this accident can only happen in the event when the wounded vessel is empty, and its walls are prevented from collapsing, and the wound remains patulous. He found these conditions normally present in the sinuses of the dura mater, the hepatic veins, the superior vena cava, the internal jugular, the subclavian, and axillary veins, because the walls of all these vessels are firmly fixed to the adjacent tissues, which prevents their collapse on being wounded. It has been further shown that the admission of air is favored by pathological conditions which affect the veins in a similar manner, as the existence of induration of their walls, the result of chronic inflammation, or infiltration by neoplasms. Experience has corroborated these views, inasmuch as it has been shown that this accident has occurred most frequently in operations in the vicinity of veins which, from their anatomical location, are prevented from collapsing by firm and unyielding layers of fascia, and vessels which are, or have become, adherent to unyielding tissues. Again, it is always very justly feared in removing tumors which have become adherent to large veins, as the morbid process frequently has impaired the normal resiliency of the vessel so as to keep its lumen patent in the event it is wounded during the dissection.

In 33 cases of intravenous aspiration of air collected by Couty,[1] air entered the external jugular 9 times, the axillary 8 times, the internal jugular 5 times, the subscapularis 3 times, the facial twice, the anterior jugular twice, the occipital twice, and twice one of the anterior thoracic veins in close proximity to the clavicle. In 1864 Greene collected 67 cases where air had entered a vein during an operation. The greater number of cases occurred during extirpation of tumors in the region of the neck, chest, and axilla. Twice the accident took place in disarticulating the humerus at the shoulder-joint (Cooper, Delpech); once on extirpating the scapula and clavicle (Mussey); twice on tying the subclavian artery (Rigaud, Clemot); three times on bleeding from the external jugular vein, three times on bleeding from the median vein, and once on passing a seton-needle through the tissues in the regions of the neck. Among the wounded veins the external jugular is mentioned 13 times, the internal jugular 10 times, the subclavian and axillary each once. In the remaining cases the vein is not specified, or the injury involved a branch in close proximity to the specified vessels.[2] All causes which interfere with the free return of venous blood prevent the admission of air; while, on the other hand, all influences which promote the venous circulation, such as an unimpaired *vis a tergo*, regular deep inspiration, and the force of gravitation predispose to this accident. Soon after Beauchène made known his case the Royal Academy of Medicine of Paris appointed a commission to investigate the subject. In the report it is stated that the results of the experiments, which were made principally on dogs, had proven that the conditions necessary to determine the entrance of air consisted in making the wound in the vein anywhere within the area of the venous pulse, or, at any rate, only a short distance from it. If the wound was located at a greater distance and beyond the influence of the venous pulsations, no air would enter, although the wound was kept open. The experiments were also to determine the extent of the venous pulse, and the conclusions arrived

[1] Op. cit.

[2] American Journal of the Medical Sciences, 1864, p. 38.

at were that the brachial and axillary veins were beyond the venous wave, while the subclavian and lower third of the jugular veins were the seat of pulsations, consequently wounds of the veins in the localities were liable to admit air. The sound produced by the entrance of air is described as resembling the lapping of a dog or cat, and that it always occurred during, and synchronous with, inspiration; but sometimes, when it was heard more frequently, it accompanied the diastole of the right ventricle. After the air had entered the vein, the sound which could be heard on auscultation over the heart was described as a "bruit de soufflet," synchronous with the action of the heart. In regard to the effect of the aspirated air it was decided that, in order to produce a fatal result, it was not only necessary that the amount of air introduced should be considerable, but that it must be thrown into the vein with some degree of force.

Hertwig called special attention to the fact that aspiration of air is not as frequent an accident as is generally supposed, and that for its occurrence it is necessary that the peripheral flow of blood to the wound must be obstructed, that the edges of the vein wound must be drawn apart, and finally, that even the introduction of a canula into the vein is necessary to admit a sufficient amount of air to produce serious results.

The first case of admission of air into a vein that was recognized and verified by a post-mortem examination occurred in the practice of Beauchène, and is described by F. Magendie.[1] As the case is of great historical and scientific interest, I will relate it as described by Magendie.

A locksmith, 23 years of age, had for five years a large tumor on the right shoulder and clavicle. His acute sufferings induced him to enter the hospital to have it removed. It was necessary in the operation to remove the middle portion of the clavicle. Thus far the success was complete; but little blood was lost, the pulse was good, and the breathing easy, when the patient suddenly cried out: "My blood is leaving my body! I am dead!" At the same

[1] Physiological Researches on Life and Death, by X. Bichât, with Notes by F. Magendie. Translated. Boston, 1827, p. 188.

moment he became stiff, lost his consciousness, and was covered with a cold sweat. A singular and rather loud noise was heard in the interior of his chest. The surgeon thought that he had opened the pleura by removing a portion of the clavicula, and thus given access to the air and to the blood to the right side of the thorax. The fingers of an assistant were immediately thrust into the bottom of the wound with a view of stopping the supposed opening in the pleura, and the surgeon endeavored to introduce into the thorax the extremity of a sound of gum-elastic. When he thought that he had succeeded he drew with his mouth the air which he supposed to be effused in the pleura. He wished then to proceed to the dressing, and, in order to do this, he substituted for the fingers of the pupil, which were at the bottom of the wound, a sponge covered with wax; but the moment the sponge took the place of the fingers, the same noise that was at first heard, and which had ceased in an instant, was renewed with more force than before. The syncope and cold sweat still continued. Water thrown into the face made him give some signs of life, but he died a quarter of an hour after the appearance of the accident I have first described, and forty-five minutes after the commencement of the operation. The body was examined the next morning. They expected to find the right pleura open, much blood and air effused into its cavity, and the lungs on that side collapsed. Nothing of the kind was found. The pleura was whole and there was no effusion in it. The lungs were as usual, but an opening of half an inch in extent was discovered in the external jugular vein, at the place where this vein opens into the subclavian. The cavities of the heart were large, but contained no blood. Bubbles of air were observed in the vessels of the brain; the other vessels were not examined.

In order to study some of the conditions under which air has been aspirated into veins, and for the purpose of ascertaining the effects of such accidents in man, I will introduce a number of well-authenticated cases which will represent a great diversity in the point of entrance, and will also aid in the establishment of the fact that this accident can occur outside of the regions of venous pulse, and always occurs during inspiration, and is never produced by the aspiratory function of the heart.

External Jugular. Barlow's case.[1] The patient was a female suffering from a tumor seated on the side of the neck, which had been increasing in size for several years; its base was extensive, and occupied the whole of the lateral and posterior parts, extending from the ear to near the sternum, and sidewise from the thyroid gland to the sterno-mastoid muscle, under which a part of the tumor was situated. The patient was seated in a reclined chair, supported by assistants. Two superficial elliptical incisions, ten inches in length, were made downwards from a little below the ear, "when on proceeding to dissect the skin aside to get at the basis of the tumor, a sudden and unexpected hissing and gurgling noise rushed obviously from a large divided empty vein, and the patient expired instantly, without either sigh, groan, or struggle, and every effort to restore animation became fruitless." The divided vein appeared larger than the normal external jugular, but the reporter believes that it was this vessel or an anomalous vessel greatly enlarged. As the incisions must have traversed the external jugular, according to his own description, it was undoubtedly this vessel which was injured. It is distinctly stated that the vessel was flabby and empty, and that the instant the atmospheric air gained access and filled the vacuum, the hissing noise ceased, the patient expired, and the mouth of the vessel collapsed.

Remarks.—In this case the admission of air was favored by the dilatation of the vein, and the semi-erect position of the patient. The latter factor produced the emptiness of the vein. The instantaneous death without any symptoms preceding it can only be explained by the fact that the air entered with force and in large quantity the right ventricle and arrested the heart's action by over-distension.

Internal Jugular. Ulrich's case.[2] The operation was performed for the removal of a tumor involving the left side of the neck. It was found that the tumor was attached to the deep vessels of the neck, and in severing its connection, the internal jugular vein was opened. No hemorrhage followed, the vessel remained open like an artery, and air entered immediately. The patient fainted;

[1] Medico Chirurg. Trans, vol. xvi. p. 29.

[2] Rust's Chirurgie, Berlin, 1836, vol. xvii. p. 565.

twitching of the muscles of the face, opisthotonos, a few slow respirations followed, and the patient was dead. The vein was found obliterated above the incision, and thickened, and more resistant than normal where the wound was inflicted. The reporter attributed the ingress of air to aspiration of the heart, and death to paralysis of this organ.

Remarks.—The pathological changes in the vein above the wound, interrupting entirely the column of blood from above as well as the thickening of the incised vein walls were potent factors which determined the entrance of air. As this operation was performed before anæsthetics were used, we may be almost certain that the patient was in a sitting or half-reclining position during the operation, thus favoring greatly venous return and ingress of air. The entrance of air in this instance is brought in connection with the suction power of the heart by the author in accordance with the prevalent doctrine of the French Commission.

Internal Jugular. Dupuytren's case.[1] The operation consisted in the removal of a tumor of a fibro-cellular character of considerable size from the neck of a female, 22 years of age. No serious obstacles presented themselves until the last deep attachment was severed with the knife when suddenly a prolonged hissing noise (*Soufflement prolongé*) was heard resembling the sound produced by the entrance of air into a vessel from which it had been exhausted. The patient immediately proclaimed "I am dying," and instantaneously dropped down on the floor, a lifeless corpse. As no other cause was found which could in any way account for the sudden death, the fatal issue was attributed to the entrance of air into the internal jugular vein. The following account of the post-mortem appearances fully warrant this supposition: "The right auricle was distended with air like a bladder, which rushed out when cut open without any admixture of blood. Fluid blood was found in the different vessels. Great quantities of air were found in all the vessels. There was no other unnatural appearance in any other part of the body."

[1] Medico-Chir. Trans., vol. xvi. p. 301.

Remarks.—The editor of the *Medical and Chirurgical Review* explained the entrance of air in this case as follows: "It proves that the heart acts as a sucking as well as forcing pump, otherwise air could never have passed from a cut vein in the neck down into the right chambers of the heart. It is highly probable that, in consequence of the morbid state of the parts, the mouth of the cut vein had remained patulous, and thus readily admitted the air." As no mention is made of the occurrence of hemorrhage, the vein was probably empty, a condition which might have been owing to the position of the patient during the operation or the pressure of the tumor. It is also reasonable to assume that, on account of the intimate connection of the tumor with the vessel, the former so altered the structure of the latter as to prevent closure of the wound, all of these causes combined resulting in aspiration of air during inspiration.

Facial Vein. Mott's case.[1] The operation consisted in extirpation of the parotid gland, the seat of a scirrhous tumor. The facial vein was opened, where it passes over the base of the lower jaw, in dissecting the integuments from the tumor in the early stage of the operation before a single artery was tied. At the instant this vessel was opened, the attention of all present was arrested by the gurgling noise of air passing into some small opening. The breathing of the patient at once became difficult and laborious, the heart's action violent and irregular, his features were distorted, and convulsions of the whole body soon followed to so great an extent as to make it impossible to keep him on the table. He lay upon the floor in this condition for nearly half an hour, as all supposed in *articulo mortis*. The convulsions ceased gradually, his mouth was distorted, and complete hemiplegia was found to have taken place; after an hour had passed he could speak, but the use of his arm and leg was only recovered completely after the lapse of a day.

Remarks.—Although not stated, it was undoubtedly true that in this instance the facial vein was enlarged, and its walls had lost their normal resiliency, thus favoring the ingress of air. This case is also of interest, as from the predominance of the

[1] Ibidem, p. 32.

cerebral symptoms it is apparent that some of the air must have passed through the pulmonary circulation and have gained access into the cerebral vessels from the left ventricle, giving rise to symptoms of cerebral embolism which disappeared as the air was absorbed.

Axillary Vein. Bransby Cooper's case.[1] The patient was a female, 19 years of age, who was the subject of a malignant tumor of the right humerus which required amputation at the shoulder-joint. The operation was done by making a double flap, the subclavian artery in the mean time being compressed against the first rib. There was no loss of blood. The subclavian artery was secured, compression being kept up, as there were small vessels which required ligation. As the operator was removing an enlarged gland from the axilla, he heard distinctly a peculiar gurgling noise, like air escaping with fluid from a narrow-necked bottle. At the same moment the patient fell into a state of collapse which threatened immediate death. The face was deadly pale, the pupils fixed and insensible to light, the pulse small and fluttering, at intervals regular, respiration hurried and feeble, and at irregular intervals attended with a sigh. The patient was placed in the recumbent position, the flaps closed, and stimulants applied; but an hour elapsed before she had sufficiently recovered to be removed from the operating room. Subsequently, when placed in bed, she maintained a constant motion of alternate flexion and extension of the right leg which continued for several days, at the same time she complained of pain, extending up the right side of the neck and head. The next day the pulse varied from 140–150 per minute, which remained the same for two days. She gradually rallied and recovered completely.

Remarks.—In this case the axillary vein was divided at a point where its walls are firmly fixed and its lumen kept patent by dense connective tissue which surrounds the vessel, a condition which predisposes to aspiration of air. Pulmonary air-emboli obstructed the passage of blood through the lungs, a circumstance which would serve to explain the rapid respiration and the accelerated action of the heart until the obstructing cause was removed.

[1] Med.-Chir. Trans., vol. xxvii. p. 41.

Axillary Vein. Courvoisier's case.[1] The operation was performed for the removal of a recurring cancer of the breast, and included the extirpation of infiltrated and ulcerated axillary glands. As the dissection reached the upper margin of the mass of axillary glands a lapping (schluerfendes) sound was suddenly heard; at the same time the patient, a robust woman 58 years of age, sank into a condition of collapse. The central portion of the vein was at once closed by digital compression, and artificial respiration with the administration of stimulants were successful in restoring her after the lapse of half an hour. Both ends of the vein were ligated, and the central ligature included the forceps, which were allowed to remain. The patient recovered.

Remarks.—In this instance the entrance of air was again determined by the anatomical location of the vein wound, to which may have been added cancerous infiltration of the paravascular tissues, which rendered the vein walls still more unyielding. The amount of air admitted must have been small, to judge from the evanescent nature of the symptoms which followed.

Anterior Thoracic Vein. Amussat's case.[2] The patient was a woman, 47 years of age, suffering from a scirrhous affection of the right mammary gland and the subjacent and surrounding tissues. The breast and adjacent tissues had been removed, and the operator was dissecting towards the opposite side, when suddenly, on making an incision into some suspicious granulations on the inner side of, and below the left clavicle, he and three other surgeons who were assisting him heard a sudden, distinct, interrupted sound, as of air passing into a cavity through a narrow opening. The patient exclaimed, "I am dying," and appeared to be suffocating. A repetition of the same sound convinced the operator that air had entered through a wounded vein, and he placed his finger on the spot from which the sound proceeded. The patient's condition became critical; a cold sweat covered her face, her eyes were turned upwards, and all around her thought her dying. The orifice of the wounded vein could be distinctly seen. The chest was compressed with a

[1] Correspondenz blatt fuer Schweizer Aerzte, p. 205, 1880.

[2] Gazette des Hôpitaux, July 6, 1837.

view to force out the air from the vein, the wounded spot being compressed during the expansion of the chest. The patient soon began to show signs of improvement, when the operation was completed, and the vein with a portion of the tissue was tied. The patient recovered completely.

Remarks.—Although the particular vein wounded in this instance is not specified, it was undoubtedly a branch of the subclavian vein, the wound being in close proximity to the latter vessel. This case furnishes a good illustration of the fact, that veins of comparatively small calibre, when wounded near their proximal termination into a large vessel, may serve as points of entrance of air under the same circumstances as when the principal trunk is injured.

Superficial Cervical Vein. Trélat's case.[1] M. Trélat related, at a meeting of the Société de Chirurgie of Paris, an important case in which sudden death occurred in a patient from whom he was proceeding to remove a submaxillary tumor. The patient turned ghastly pale, and the heart's action ceased suddenly. Artificial respiration and electrization of the phrenic nerve induced some respirations and a slight return of color after fifteen minutes, but ineffectually. At the post-mortem examination a small vein opening into the external jugular was found to have been partially divided; in the jugular was a long clot interspersed with air-bubbles, and other bubbles of air were found in one of the mediastinal veins and the posterior cardiac vein, and a very notable quantity of air in the right chambers of the heart.

Remarks.—Several members of the society argued that death in this instance was due to the anæsthetic, and not to the entrance of air into the vein. Roux and Giraldès claimed that in several cases of death from chloroform they had found gases in the heart, in the vena cava, and even in the veins of the pelvis, but M. Depaul, in reply, properly and forcibly pointed out that the air in this case occupied only the veins going to the heart and the wounded vein.

[1] British Medical Journal, March 16, 1872.

Femoral Vein. The only well-authenticated case of aspiration of air into the femoral vein that I have been able to find is recorded in the *Medical and Surgical History of the British Army in Turkey and the Crimea*, vol. ii. p. 277, and refers to the sudden death from this cause of a soldier who had suffered amputation of the thigh. Three days and one-half after the operation he died suddenly without any obvious cause. At the necropsy, twelve hours after death, it was ascertained that the right cavities of the heart were distended with a mixture of blood and air, and the same condition was found in the two iliac veins and the inferior vena cava.

Remarks.—As the time which had intervened between the operation and the fatal accident was more three days, it is necessary to assume that the venous thrombus had been removed by suppuration, thus opening the vein for the admission of air, or that the supposed air found in the heart and vessels was not air but gas which had developed in the wounded parts, and had gained entrance into the venous circulation. This latter supposition is strengthened by the statement that the surfaces of the flaps were separated by gaseous products, and that the femoral vein was not closed but lay open on the surface of the stump.

Internal Saphenous Vein. Warren's case.[1] The operation was done for the removal of a tumor from the inner surface of the thigh. In the dissection the internal saphenous vein was wounded; the event was promptly announced by an audible and distinct sucking sound produced by the entering air. No alarming symptoms followed, as the further ingress of air was promptly prevented by closure of the vein.

Uterine Veins. That the entrance of air into the uterine veins might be a cause of danger after parturition was suggested by Legallois in 1829. Dr. John Rose Cormack read a paper on this subject before the Westminster Medical Society in 1850 when he gave the details of three cases that had occurred in his neighborhood. Many authorities doubt the possibility of admis-

[1] Gazette Médicale, No. 52.

sion of air into the uterine veins after labor. Julius M. Kolb[1] alludes to this subject as follows: "I have not seen a case which convinced me that air had passed into the open veins of a recently delivered uterus, and I cannot conceive the mechanical possibility of such an occurrence." Bessems, Lionet, Lever, Wintrich, Berry, and Simpson assert that they have met with such instances, and a sufficient number of well-authenticated cases have been placed on record which leave no further doubt as to the possibility of sudden death in puerperal women from entrance of air into the uterine sinuses. In a recent number of the *Wiener Medicinische Zeitschrift*, Braun gives three fatal cases from the introduction of air into the uterine veins; in two of three the uterine douche was used, in one to produce abortion, in the other after delivery of twins, and the patients died in a short time, one indeed within twenty minutes. Post-mortem examination showed air in the uterine veins, in the ascending vena cava, and in the veins of the heart. The third case was that of a woman who had been delivered lying upon her left side, and was then turned upon her back; massage was made over the uterus, she gasped and died in a few minutes. Braun suggests that in the change of position a volume of air entered the uterus, and the manipulation, instead of driving it out, forced it into the uterine veins. Bischoff refers to two cases that came under his observation.[2] Dr. Draper[3] has reported two cases where instant death occurred from efforts to cause criminal abortion. The post-mortem examination proved in each case the presence of air in the veins.

Mr. George May[4] reports three cases which occurred in his vicinity. The patients died respectively immediately, six hours, and eight days after delivery, and in all of them post-mortem examinations showed the presence of air in large quantities in the inferior vena cava and the right side of the heart. An inte-

[1] The Pathological Anatomy of the Female Sexual Organs, 1868, American Translation.

[2] Corresp. bl. f. Schweizer Ärzte, p. 206, 1880.

[3] Boston Med. and Surg. Journal, January, 1883.

[4] British Med. Journal, June 6, 1857.

resting account of this accident is given by Dr. George Cordwent, and relates to a case that came under his observation.[1]

His patient was 28 years of age. During the delivery, at full term, her expulsive pains became urgent, and at her request she was permitted, in the absence of her medical attendant, to remain standing; after a few severe pains the child was expelled, and after falling on the floor dragged with it the whole placenta. Almost immediately afterward a kind of gurgling sound was heard by the attendants, but whether it arose from rumbling in the bowels they could not say. The patient remained about one minute standing as before and holding on to the bed-post; she then cried out: "I can't see! I feel faint! lay me on the bed," and expired almost instantly. At the necropsy, 24 hours after death, it was shown that the uterus externally presented the normal appearances of a recently delivered organ, except that a portion of the wall of its fundus to about the extent of a five-shilling piece was slightly more puffy than the other portions, and, on cutting into it, air-bubbles escaped. There had been no laceration of the placental surface; the uterine cavity contained only one small clot; its lining membrane was healthy. The coronary vein of the stomach was distended; the right side of the heart was slightly gorged, and when the auricle was punctured air-bubbles escaped with the blood which it contained.

Davidson[2] reports the case of a Hindoo woman who was admitted into the Kaira Gaol Hospital and safely delivered of a female child. The labor was in every respect normal. The placenta came away at the usual time, and there was no post-partum hemorrhage. About three-quarters of an hour afterward the woman died without any apparent cause. There had been no hemorrhage or convulsions. The patient had been taking some nourishment when she suddenly fell back and expired. At the post-mortem examination two hours after death the uterus was found empty, with large and somewhat distended veins; the right side of the heart contained a quantity of air mixed and churned up with blood, which escaped in bubbles,

[1] St. George's Hosp. Reports, vol. vi.

[2] The Lancet, vol. i. p. 999, 1883.

the lungs were congested; all the other organs were normal. The most interesting and convincing case is related by Olshausen. It is the most convincing on account of the painstaking and accurate post-mortem examination which was made to determine the cause of death.[1]

A robust secundipara, aged 29, was delivered at full term. The uterus was unusually distended; no albumen in urine. The labor was lingering and the uterine douche was used. The water was of 30° R. and was forced into the vagina gently by a pump. A third injection was made by a midwife. After eight minutes' use the patient began to complain of oppression. The tube was withdrawn. The patient rose in bed, immediately fell back senseless, and died in less than a minute under convulsive respiratory movements and distortion of the face. Eight minutes later bleeding by the median vein was tried, but only a few drops flowed. On touching the body distinct and widespread crepitation was felt.

Autopsy eight hours after death.—A large quantity of dark fluid blood escaped from the sinuses of the dura mater. The cerebral membranes very hyperæmic; brain normal, lungs somewhat congested, heart lying transversely, apex in fourth intercostal space. Left ventricle in firm contraction, right quite soft, something like an intestine with thick walls; the coronary vessels contained a quantity of air bubbles. Left heart contained only a small quantity of blood; the right held little, but it was frothy. The distended uterus crepitated everywhere on pressure under the hand. A number of subperitoneal vessels of medium size were plainly filled with air. The right broad ligament was strongly distended with air bubbles, and this emphysema of cellular tissue extended from the broad ligament through the retro-peritoneal space to the inner side of the right kidney, and even below the liver to the inferior vena cava. *The inferior vena cava was enormously distended*—it was at least an inch in diameter—containing mostly air. The uterus was divided in the median line; a placenta was attached to the anterior wall; a small flap was detached from the uterus, a second placenta was attached behind and to the right; a larger portion of this had been separated, so that there was a sort of a pouch between it and

[1] Monatsschrift f. Geburtskunde, Jan. 1865. American Journ. Med. Sciences, July, 1865.

the anterior wall. The two ova were uninjured. The air had gained access into the veins at the placental site. It was concluded that the tube had been passed into the uterine cavity and that air had been thrown in with the water by the pump.

It would be difficult to conceive in what manner air could be drawn into the uterine veins by the aspiratory movements of the chest or heart, as is the case in the veins about the apex of the chest. Another explanation must be sought for, and this will be found in the change of structure, and the relations of the uterine veins. The veins during pregnancy keep pace with the enormous physiological hyperplasia of the uterine tissues, and are gradually converted into large sinuses, more especially the vessels at the placental site; they are simply excavations or channels in the contractile muscular walls of the uterus, their size being subject to the state of the uterine walls, whether at rest, relaxation, or contraction. When the placenta is detached, some of these sinuses are laid open, and in a normal condition their calibre is obliterated by the contractions of the uterus and the formation of thrombi. If, from any cause, air should reach the uterine cavity, it may be aspirated into the uterine sinuses by relaxation of the uterine contractions, and, having gained access into them, it is readily forced into the circulation by subsequent contractions; the uterine walls acting the part of a suction and forcing-pump. During forcible uterine contractions the veins are nearly emptied of their contents, and, as the organ relaxes, the walls of the veins are distended and a vacuum is formed, which is filled with blood or air. Should the relaxation be slow, the empty spaces are readily filled with blood or serum in the absence of air, but if the uterus relaxes quickly the suction power is proportionately greater, and, in the event air has reached the uterine cavity, it is aspirated into the open veins, and, by reaching the right side of the heart through the vena cava, it gives rise to the same train of symptoms as when it is admitted into a vein during a surgical operation in the regions of the neck.

Pulmonary Vein. Dumin's case.[1] This is the only case on record where it is claimed that death was produced by the entrance of air from a pulmonary tubercular cavity through the pulmonary vein into the left side of the heart. The patient was a young man suffering from pulmonary tuberculosis in the last stage. Physical diagnosis revealed a large cavity in the apex of the right lung. After the patient had been in the hospital for three weeks the general conditions remained about the same, while the local destructive process had been progressing. One day, after eating his dinner, he arose from his bed, fell down, and expired almost instantly without uttering a word or sound. At the post-mortem examination, twenty-four hours after death, it was found that the apex of the lung contained a cavity of considerable size, besides extensive crude infiltrations. The left lung contained numerous nodules and three small cavities. The third cavity in the substance and near the base of the lung contained a small amount of blood intimately mixed with air-bubbles. The heart was slightly dilated. The left ventricle was filled with blood mixed with innumerable small air-bubbles. The right cavity also contained air, but in much lesser quantity. All the larger arteries contained air mixed with blood; air-bubbles were also found in the venæ cavæ and the pulmonary artery. The arteries and veins in the brain and meninges were found distended almost exclusively with air. No signs of advanced putrefaction could be found, and none of the parenchymatous organs contained gases. The reporter explained the sudden death by the entrance of air from the small cavity in the left lung, which contained spumous blood, the air having found its way into an open branch of the pulmonary vein, and from thence into the left side of the heart. The air, which was found in the right side of the heart and veins, according to his view, had passed through the systemic capillaries. As the direct cause of death, anæmia of the brain is mentioned.

Remarks.—It seems to me that several reasons might be mentioned which would throw doubt on the correctness of the assertion that, in this case, the immediate cause of death was owing to entrance of air into the pulmonary vein. 1. The time which had elapsed from the commencement of the attack until

[1] Berliner Klin. Wochenschrift, January 30, 1882.

death took place was not sufficient to produce such an extensive distribution of air, unless it could be proved that the heart's action continued after respiration had ceased. 2. The existence of an open vessel in any of the cavities was not proven at the examination after death. 3. The body appears to have been affected by a certain amount of putrefaction, which may have been sufficient, in degree, to give rise to the evolution of gases, and the putrefactive changes may have been limited to, or were at least farthest advanced in, the blood, which would explain the absence of gas in any other part of the body, except within the bloodvessels. 4. Snycope is a frequent cause of sudden death in greatly debilitated patients when the heart is called upon to perform an increased amount of labor, as when the patient suddenly assumes the erect position.

Superior Longitudinal Sinus. Volkmann's case.[1] The only fatal case of admission of air into the sinuses of the dura mater is reported by Genzmer. The patient was a female, 63 years of age, who was affected with a perforating sarcoma of the dura mater. The tumor was noticed about two years before the operation, and was located in the region of the posterior extremity of the sagittal suture, and for a long time gave rise to no inconvenience. For the last six months it caused intense headache. On one occasion, a physician believing that it was an atheroma, attempted its removal, but as the first incision gave rise to copious hemorrhage, he desisted from any further attempts, and the wound healed kindly. When the patient was admitted under Volkmann's care into the Clinic at Halle, the tumor presented a lobulated appearance, being composed of three parts, each about the size of a plum, and was located over the posterior extremity of the sagittal suture. On touch, the tumor was soft and elastic, and imparted to the finger distinct pulsations. Gradual compression reduced its size one-half; when the pressure was discontinued it resumed its further dimensions. On auscultation, a blowing sound was heard synchronous with the radial pulse. By pressing the end of the index finger deeply between the lobes of the tumor, a bony defect in the skull was readily detected. The conclusion was reached, that the tumor had sprung from the dura

[1] Verhandlungen d. Deutschen Gesellschaft f. Chirurgie, vol. vi. p. 32.

mater, and had perforated the skull by the prolonged pressure, causing interstitial absorption of the cranial vault. During the patient's stay in the hospital the tumor increased very rapidly in size. As no brain symptoms were present, it was assumed that the substance of the brain was intact. In view of the speedy fatal issue, which of necessity would take place without operative interference, Volkmann decided to remove the tumor. The operation was done April 2, 1875. Under strict antiseptic precautions the tumor was exposed by a crucial incision, and the flaps reflected with the periosteum to the margins of the opening in the skull. The aperture in the bone measured $5\frac{1}{2}$ by $4\frac{1}{2}$ cm. in diameter. With a Luer's cutting forceps the opening was enlarged to 7 by 8 cm. The tumor, when exposed, was nearly as large as a fist, and firmly adherent to the dura mater. The dura mater was carefully divided around the margins of the tumor, which had now been liberated from all its attachments except the falx cerebri. It was now drawn forward through the opening in the skull, and the falx cerebri divided with scissors from before backwards. This step of the operation was attended by alarming hemorrhage. As the blood was being sponged away to expose momentarily the field of operation, a peculiar and characteristic lapping sound was heard, which indicated to all present that air had entered the longitudinal sinus. At the same time the assistant, who was giving the chloroform, remarked, "She is dying." The wound was immediately compressed with a large carbolized sponge. The patient was in collapse, her breathing was interrupted and stertorous. After a short pause, it was determined to complete the operation, but as soon as the tumor was again drawn forward, and its attachment at the junction of the longitudinal with the transverse sinuses was divided, air again entered, accompanied by the same characteristic sound. The tumor was separated rapidly from its remaining attachments, and a Lister dressing was applied in such a manner as to make at the same time a requisite amount of compression for the double purpose of arresting hemorrhage and preventing further ingress of air. At this time the patient was pulseless, pupils dilated, extremities cold and blue. Auto-transfusion, by constricting the arms and legs with elastic bandages, had the effect of momentarily stimulating the heart, but respiration became more irregular and interrupted, and after a few more brief moments the patient died. At the post-mortem examination, which was held on the following day, the right side of the

heart was opened under water, air-bubbles escaped, showing conclusively that air had made its entrance through the longitudinal sinus. The left side of the heart contained no air. Air was also found in the pulmonary artery and the subpleural vessels. The left side of the brain had suffered more from compression by the tumor than the right. The defect in the dura mater corresponded to the opening in the skull. An additional source of hemorrhage was detected at the posterior margin of the defect in the cranium, where the opening of a vein in the substance of the bone, 5 mm. in diameter, could be seen. Under the microscope the tumor showed small spindle-shaped cells, with a very vascular intercellular substance.

Remarks.—In this case all circumstances favored the entrance of air into the wounded sinus. The sudden and severe loss of blood from such a large reservoir as the longitudinal sinus rendered the vessel empty, thus creating the most essential element in the causation of air aspiration. The position of the patient during the operation undoubtedly was such that the force of gravitation assisted materially in the formation of a vacuum. The walls of the sinus being rigid and attached to the surrounding structures prevented collapse of the vessel, and held the wound patulous. That death was owing to the introduction of air is sufficiently proven by the symptoms during life and the evidences derived from the post-mortem examination.

Veins of Diploë. Franck[1] asserts that he has repeatedly seen aspiration of air into the veins of the diploë after trephining. He claims that the air reaches the heart through the medium of the vertebral veins, which, from their protected position, are more favorably located for this purpose. By experiments he proved that ligation of the jugular veins does not prevent the aspiration of air through the veins of the diploë, while, on the other hand, this accident cannot happen when the vertebral veins are compressed. As the veins of the diploë in some instances are unusually large, and their walls firmly attached to

[1] Sur la transmission de l'aspiration thoracique jusqu' aux canaux veineux des os du crâne, etc. Gazette Méd., No. 25, 1881.

the unyielding bone tissue they constitute channels which cannot contract in the event they are injured, consequently we should *a priori* expect that aspiration of air will take place under the same circumstances as in the case of the sinuses of the dura mater, and in all extensive injuries of the cranial bones the same caution should be exercised to guard against this accident. In troublesome hemorrhage from venous sinuses in bone, the bleeding is promptly and safely arrested by implantation of an aseptic sponge, which can be left *in situ*, as it will be removed by the granulation tissue during cicatrization. In such instances the sponge is peculiarly well adapted, as the lumen of the vessel is surrounded by unyielding bony walls, which will support any amount of pressure on part of the aseptic tampon.

XI. *Experiments on Venous Air-Embolism.*

The injection of air was always made into the jugular vein. The neck was shaved, and the surface disinfected with a five per cent. solution of carbolic acid. Ether was always used as an anæsthetic, the animal being kept fully under its influence until everything was in readiness to thrown in the air, when the inhalation was suspended, and the animal was allowed to come out from under its influence, for the purpose of studying the effects of the air on the heart and respiration, independently of the effects of the anæsthetic. The vessel was freely exposed, usually in the lower part of the neck, by a parallel incision. After isolating it to the extent of from two to four inches, the influence of the respiratory movements of the chest on the venous circulation was carefully studied. Then a hæmostatic forceps was applied to the distal portion of the vein. Below the point of compression the blood was forced out of the vessel between two fingers, and its return prevented by applying another pair of forceps to the proximal end of the exposed vein. We had thus a bloodless portion of vein between the forceps, presenting a ribbon-like band. This was partially divided in an oblique direction for the purpose of facilitating the introduction of a canula. The canula was securely fastened in the vein by a

ligature, when the proximal pair of forceps were removed, and, by compressing the bulb, the air was injected with force, so as to imitate as nearly as possible the conditions present during the accidental introduction of air. The canula was connected with a rubber-bulb, of known capacity, by means of a rubber-tube. After the completion of the experiment (if the animal survived) the vessel was divided completely, and both ends ligatured with catgut, and the wound closed with a continued catgut suture. The weight of the animal and amount of air injected were estimated accurately in most instances.

Experiment No. 1.—Sheep, weighing 120 pounds. Left jugular vein. The vessel was opened in its lower third, but no air entered. A rubber-tube was introduced for a distance of two inches with a view of facilitating the spontaneous ingress of air, but this accident failed to occur. Air was injected, at intervals of eight minutes, in quantities of 30 c. cm each, until the enormous amount of 480 c. cm. had been introduced. After the first injection nothing was observed that indicated the presence of air in the veins or the heart. After the second dose a slight splashing sound could be heard over the cardiac region, which became louder and more distinct as the amount of air in the right side of the heart increased. The first serious symptoms observed were a tumultuous action of the heart and difficulty in breathing, which became aggravated by every succeeding injection. Towards the end of the experiment, which lasted nearly two hours, the animal was attacked, at short intervals, by general convulsive movements. After the suspension of respiration the heart's action became very slow and feeble, and at times irregular. The immediate cause of death was plainly due to asphyxia, as manifested by the great dyspnœa and the cyanotic hue of all visible mucous surfaces. On examination after death, a few air-bubbles and only a small amount of dark blood were found in the left ventricle. The right ventricle was arrested in the diastole and contained a large quantity of very dark, almost black, spumous blood. Air-bubbles were found in a number of distant arteries of small size.

Experiment No. 2.—Adult, large cat. In this instance the canula was introduced and tied in the left jugular vein. The heart was

exposed before the injection was made with a view of observing directly the effects produced by sudden inflation of the right cavities of the heart. Before the air was introduced, the heart contracted regularly—artificial respirations being made for the purpose of preventing death by asphyxia. As soon as the right side of the heart was distended by the air, the left auricle and both ventricles ceased to contract, while the right auricle continued to pulsate. The pulsations were feeble and irregular. The coronary veins became filled with air-bubbles, presenting the appearance of a rosary. On opening the superior vena cava, air and frothy blood escaped, the right side of the heart collapsed and all chambers of the heart commenced to contract regularly and with considerable force. The pulsations continued for 15–20 minutes, becoming more feeble and irregular and intermittent towards the last. After death, air was found in both venæ cavæ and the iliac veins. The left ventricle was completely empty. In this case, owing to the small size of the heart and the large amount of air introduced, the contractions of the right ventricle were arrested in the diastole, while respiration continued. Death took place suddenly from mechanical over-distension of the heart.

Experiment No. 3.—Dog, weight 65 pounds. Injected 30 c. cm. of air into left jugular vein. Churning sounds over cardiac region loud and distinct. Heart's action became very tumultuous and intermittent. Respirations superficial and rapid. The animal was bled from the distal end of the jugular vein to the amount of four ounces, whereupon the heart's action became regular and the respirations diminished in frequency. The vein was divided completely and both ends were tied with fine catgut ligatures, the wound being closed in the usual manner. For a number of days the dog appeared quite unwell, showed no disposition to eat and acted very stupidly, being inclined to sleep most of the time. Subsequently he recovered completely. In this case the intravascular pressure was promptly relieved by free bleeding which enabled the heart to force the air through the pulmonary into the general circulation. The stupid condition of the animal was undoubtedly owing to embolism of the cerebral vessels, which disappeared after the disappearance of the air-emboli by absorption.

Experiment No. 4.—Adult, medium-sized cat. Injected 15 c. cm. of air into left jugular vein. Heart's action arrested at once.

Respirations which were irregular ceased a few moments later. The chest was opened at once. The right side of the heart was found enormously dilated and almost motionless. Coronary veins filled with air. The right ventricle was punctured with the needle of an aspirator and its contents withdrawn, when the pulsations were re-established. The ventricle was again inflated through the needle of the aspirator. Five minutes after this injection the pulsations numbered about 250 per minute. Five minutes later the left auricle ceased to contract, the movements of the right being irregular and about 80 to the minute. After the lapse of another five minutes the pulsations of the ventricles were only 17 a minute and a little later all movements ceased. This experiment demonstrates that the arrest of the heart's action was due to mechanical over-distension, as aspiration of the right ventricle was followed by regular and strong contractions in all cavities of the heart. The contractions were not the result of the mere mechanical irritation of the heart by the puncture, as other and equally severe irritants were previously applied without producing any effect.

Experiment No. 5.—Dog, weight 35 pounds. Before operation, respirations 40, pulse 140. Injected 20 c. cm. of air into right jugular vein. Convulsions followed which lasted for about two minutes. Respiration rapid and stertorous. Pulse 300. After five minutes the animal made repeated attempts to get up and walk, but invariably fell down on account of imperfect control over the movements, or paralysis of the posterior extremities. Half an hour later the animal was able to walk, but appeared very feeble. Pulse 120, respirations somewhat accelerated. Recovery was complete. In this case the equilibrium of the circulation was soon restored and the air in the right side of the heart passed through the pulmonary into the general circulation in a very short time, as was evident from the presence of symptoms indicative of embolism of some of the vessels in the cerebro-spinal centres.

Experiment No. 6.—Dog, weight 75 pounds. Injected 60 c. cm. of air into the right jugular vein Churning sounds loud and distinct; heart's action labored. Respirations exceedingly rapid, later stertorous. The animal recovered rapidly from the immediate effects of the air-embolism, and was soon as well as before the operation.

Remarks.—These experiments tend to prove the following statements: 1. A small amount of air in the right side of the heart in a healthy animal gives rise only to temporary symptoms referable to the heart's action and the pulmonary circulation. 2. When air has been introduced into the right side of the heart in such quantities as not to arrest the contractions of the heart at once, it is forced through the pulmonary capillaries into the left side of the heart by the contractions of the right ventricle. 3. The danger attending the insufflation of air into veins is proportionate to the amount of air introduced, as well as to the capacity of the right ventricle to resist intra-cardiac pressure. 4. When a fatal dose of air has been introduced into the venous circulation, death takes place almost instantaneously from arrest of the heart's action, or later from suffocation. 5. Spontaneous ingress of air into a wounded, healthy jugular vein never occurred in these experiments and must be considered almost a physical impossibility, as the resilient walls of the wounded vein collapse readily when exposed to atmospheric pressure.

Dogs weighing about 30 pounds would usually recover in a short time after an injection of 30 c. cm. of air, while double that amount constituted generally a fatal dose. Sheep required a proportionally larger dose to produce a fatal result. These experiments also tend to prove that animals of the same kind and species do not manifest the same degree of tolerance to the presence of air in veins. Young animals succumb more readily to its effects; in the adult animal the degree of tolerance depends on the development and contractile power of the right ventricle. In all cases where life was prolonged for a considerable length of time after the injection of air was made, and the ventricular contractions were not much impaired, bubbles of air were found in the left side of the heart, showing conclusively that the air must have passed from the right ventricle through the pulmonary capillaries. In experiments Nos. 3 and 5 the well-marked cerebral disturbances were undoubtedly produced by secondary air-embolism of the vessels of the brain and spinal cord, since these disturbances disappeared after the removal of the emboli by absorption. When the same amount of air was administered in

one dose, it always proved more dangerous than when injected in divided doses, which simply means, that during the interval between the injections sufficient time had elapsed for the right ventricle to force at least a portion of air through the pulmonary capillaries into the general circulation; thus preventing for a time at least a fatal degree of intra-cardiac pressure. The greater the development of the right ventricle, the greater the tolerance of the animal to the presence of air in veins. This statement is well exemplified by the fact that in horses a larger amount of air is required to produce death, while in dogs a proportionately much smaller dose will result in death. This is because in the former animals the right ventricle, from its proportionately greater strength, is more competent to perform an additional task. If death followed immediately after the injection, the dose was usually a large one, the heart's action having been suspended before respiration ceased, and on post-mortem examination the right ventricle and auricle were always found over-distended and tympanitic, containing a moderate amount of blood not intimately mixed with a large amount of air, while the left side of the heart was nearly or completely empty. If death occurred some time after the injection, great dyspnœa was observed, lividity of the mucous membranes, tumultuous action of the heart, and respiration ceased prior to cessation of the action of the heart, showing conclusively that death took place from carbonic acid intoxication. In these cases the necropsy revealed a lesser degree of distension of the right chambers of the heart, which invariably contained spumous blood or blood intimately mixed with air. The pulmonary artery and its branches were filled with air and spumous blood, and bubbles of air and a small quantity of blood were present in the left ventricle. In opening the jugular vein I have always noticed that the vessel would promptly collapse as soon as hemorrhage ceased, being transformed into a pale, round cord, in which the wound could be found only with great difficulty. Air was never seen to enter, even if the wound and a portion of the vein were kept patent with forceps or a rubber-tube. I frequently arrested the column of blood in the vein by applying a compressing forceps

above, and opening the vessel below, thus creating a favorable condition for the spontaneous entrance of air, but the result remained the same. It is therefore only reasonable to assume, that air will enter spontaneously only in case a vein near the heart is wounded, the walls of which are prevented from collapsing by fixations of its tunics to adjacent, unyielding structures, or in case the vein walls themselves have lost their resiliency by previous pathological changes.

XII. *Experiments on Arterial Air-Embolism.*

Experiment No. 1.—Medium sized dog. The left carotid artery was exposed and isolated, and two hæmostatic forceps applied about two inches apart. The artery was divided between them, and the proximal end secured by a ligature. Into the distal end the canula of the injecting bulb was introduced and fastened with a ligature. When the animal had fully recovered from the anæsthetic, the forceps were removed, and 80 c. cm. of air were injected at once and with considerable force; the canula was then removed and the artery ligated. The animal collapsed almost instantaneously, and respiration was suspended for nearly two minutes. The animal appeared motionless and in a condition of profound stupor. Heart's action tumultuous and very rapid. Two minutes after the injection churning sounds were audible over the cardiac region. When respiration was re established the movements of the chest were slow and irregular, gradually becoming slower and slower until, after about fifty attempts, they ceased entirely. The limbs were rigid, and the trunk in a position of opisthotonos. The heart continued to contract for about two minutes after respiration had ceased. All vessels leading to and from the heart were carefully ligated, and the organ removed and examined under water. The coronary veins contained bubbles of air. All cavities of the heart contained air, the largest amount being found in the left auricle. Coagula were present in all of the chambers, except in the left auricle. The circle of Willis was distended with air, as were many of the smaller vessels of the brain and its membranes. The cerebral vessels were gorged with dark blood. The air injected into the carotid artery had evidently passed directly through the cerebral capillaries into the venous circulation, giving rise to venous and general air-embolism. In the

beginning respiration was immediately suspended from temporary suspension of innervation, but was re-established as soon as the amount of air in the cerebral vessels had lessened sufficiently to allow a better blood supply. It, however, ceased definitely under symptoms expressive of extensive embolism of the pulmonary artery.

Experiment No. 2.—Sheep, weight 90 pounds. Left carotid artery prepared as in previous experiment, only that the air was injected in a central direction, towards the heart. After the animal had recovered almost completely from the effects of the anæsthetic, 150 c. cm. of air were thrown in, and the proximal end of the artery was tied. The animal was at once thrown into a tetanic state, with rigid limbs and retracted neck. All reflex movements and sensations were completely suspended. The heart's action was irregular, tumultuous, and over the cardiac region distinct churning sounds were heard. Respirations exceedingly rapid. The mucous membranes accessible to sight presented a strikingly pale and anæmic appearance. Death occurred in fifteen minutes, respiration ceasing first. Examination immediately after death showed the right ventricle greatly distended and tympanitic on percussion. It contained spumous blood, air, and a few small coagula. Left ventricle contained only a minute quantity of spumous blood and fine blood clots adherent to the endocardium. Air-emboli were found in almost all vessels throughout the body. Coronary arteries distended with air. Jugular veins contained more air than blood. Basilar artery and superior longitudinal sinus contained a large amount of air. The tongue and other distant organs extremely anæmic. Although this large quantity of air was thrown directly into one of the large arteries, it was forced into the smallest vessels and through the capillaries into the veins and the right side of the heart by the powerful contractions of the left ventricle, giving origin to a combination of symptoms arising from arterial and venous embolism. Death was finally produced in a similar manner as if air had been injected directly into the veins. Some of the air had passed all the capillaries and was found in the left side of the heart.

Experiment No. 3.—Medium sized adult cat. With a view to ascertain how soon air would pass through the capillary vessels after injecting it into the carotid artery, the jugular vein on the opposite side was opened before the injection of air was made.

About 15 c. cm. of air were thrown into the carotid artery in a peripheral direction. The animal was immediately seized with convulsions and died in less than a minute. Respiration ceased first. Air was seen to escape from the wound in the jugular vein, and was also found in nearly all of the vessels throughout the body. On opening the chest the right side of the heart was found distended to its utmost, and tympanitic on percussion. The contractions of the right auricle were twice as rapid as the ventricular contractions. The movements of the left auricle were the same in frequency as the ventricular pulsations. The left auricle ceased to contract seven minutes after death. The right ventricle was punctured with the needle of an aspirator, and its contents removed, whereupon the contractions became much stronger.

8 minutes after death ventricles contracted 44 times a minute.

10	"	"	"	"	"	30	"	"
12	"	"	"	"	"	16	"	"
18	"	"	"	"	"	16	"	"
29	"	"	"	"	"	6	"	"

Then all contractions ceased and could not be restored by any kind of irritation.

Right auricle ceased beating ten minutes after death, but commenced to contract again nineteen minutes later, contracting 24 times a minute, the movements becoming more rapid and at times irregular for forty-eight minutes after death.

Experiment No. 4.—Dog, weight 35 pounds. Into the left carotid artery 60 c. cm. of air were injected in a proximal direction. The animal was seized at once with convulsions of a tetanic character with the limbs extended and rigid. Involuntary discharges from bowels and bladder. Sensation, motion, and reflex actions suspended. Pupils contracted. Respirations very rapid. Heart's action slow and labored. The left jugular vein was opened and four ounces of blood were abstracted with the result of greatly improving the heart's action. Both vessels were ligated and the wound closed. The animal was in profound stupor, resembling complete anæsthesia, until death occurred two and a half hours after the operation. Half an hour after the injection the pulse was 74, respirations 44 in a minute. On opening the abdomen the large veins were found very much dilated and contained air. Large air bub-

bles were also found in the pulmonary artery, internal mammary, and coronary arteries. Small thrombi were found in many of the ultimate branches of the pulmonary artery. The right side of the heart was distended with spumous blood and air. The left ventricle was almost completely empty, the endocardial lining presenting many patches of subserous ecchymoses. Nearly all vessels of the brain and the longitudinal sinus contained air. Cerebral and meningeal vessels engorged with blood. The animal died with marked symptoms of asphyxia. The tetanic rigidity of the extremities and the muscles of the trunk, as well as the remaining prominent cerebral symptoms, were produced by the intense cerebral congestion, the result of obstruction of some of the smaller vessels by air-emboli.

Experiment No. 5.—Adult dog, weight 20 pounds. Into the peripheral end of the right carotid artery 20 c. cm. of air were injected after the animal had completely recovered from the anæsthetic. The dog collapsed almost immediately and fell to the floor, perfectly unconscious, with extended and rigid limbs. Breathing exceedingly rapid, but gradually growing slower as consciousness returned. When the animal attempted to walk it staggered, and frequently fell, having apparently lost the power of co-ordination in the posterior extremities. The pupils, at first dilated, later became very much contracted. After about two hours, the animal walked without difficulty, but respiration, as well as the heart's action, continued very rapid. The next day complete recovery had taken place. Aside from the cerebral and spinal symptoms, the arterial embolism in this case resulted in no serious consequences; the most threatening symptoms were referable to venous embolism, showing that most of the air had passed through the capillaries into the venous circulation and right side of the heart.

Experiment No. 6.—Adult dog, weight 22 pounds. Right carotid artery exposed and 45 c. cm. of air injected towards the heart. Involuntary discharges from bladder and rectum. Pupils dilated, later contracted. Animal completely unconscious, anterior limbs extended and rigid, slight opisthotonos, breathing mostly abdominal. Churning sounds over heart heard a few seconds after injection, disappeared after eight minutes. Respiration ceased five minutes after the air was injected, but was again restored by artificial respi-

ration and faradization of vagus and diaphragm. Twenty-three minutes after the injection, pulse 128, respirations 24; seventeen minutes later, pulse 105, respirations 26; fifty-two minutes after injection, pulse 120, respirations 20; ninety minutes after injection, pulse 180, respirations 20. At this time the animal made several unsuccessful attempts to rise, but relapsed into a comatose state, which continued until death fifteen hours after the operation. At the post-mortem examination the arteries contained a good deal of dark, almost venous blood interspersed with bubbles of air. Air was found in the uterine, ovarian, iliac and mesenteric arteries and aorta; also in both venæ cavæ, pulmonary artery and veins. Some of the smallest branches of the pulmonary artery were obstructed by thrombi. Right ventricle distended with very dark, almost black, frothy blood. Left ventricle firmly contracted and almost empty. Left auricle contained dark, spumous blood. Coronary arteries distended with air; internal mammary arteries and veins also contained it. Air was found in all the cerebral vessels and sinuses which were distended with dark fluid blood. Vessels of spinal cord were intensely congested and contained numerous air-bubbles. The mucous membrane of the stomach presented eleven points of extravasation, the largest being circular and one inch in diameter. This case presented the most diffuse form of embolism, the emboli being found equally numerous and diffuse in the arterial and venous systems. The multiplicity and diffusion can be satisfactorily accounted for when we take into consideration the large amount of air which was injected, and the length of time which had elapsed from the time of insufflation until death occurred. The post-mortem appearances plainly indicated that death was produced by slow asphyxia. The primary unconsciousness and stupor were induced by the acute cerebral ischæmia; then a short period of consciousness returned as soon as the collateral circulation in the brain had become partially established, when again the animal became comatose from carbonic acid intoxication. The abstraction of blood relieved the threatening symptoms attending the arterial embolism, but failed in preventing death from asphyxia by secondary venous embolism.

Experiment No. 7.—Dog, weight 26 pounds. Injected 15 c. cm. of air into the right carotid artery in a peripheral direction. The animal had a slight convulsion and remained unconscious for about twenty minutes, when it rallied and was able to walk about the room

with a slightly staggering gait. No complications followed the operation. In this instance the cerebral symptoms followed the insufflation instantly, but owing to the small amount of air injected, they were only of short duration, the air—passing the capillaries in a short time—entered the venous circulation where it produced temporary disturbances in the pulmonary circulation.

Remarks.—Injection of air into arteries produces well-marked and characteristic symptoms which point directly to a disturbance in the circulation of the brain and spinal cord. The most prominent symptoms are: Convulsions, coma, tetanic rigidity of the limbs and extensor muscles of the back. The coma resembles complete and profound anæsthesia. These symptoms follow the operation more quickly if the injection is made in a peripheral direction toward the brain. If the animal does not succumb to the primary effect of the air upon the brain and medulla oblongata, a series of symptoms succeed which announce the arrival of air in the veins and right side of the heart. All of the experiments show that it only requires a few seconds for the powerful contractions of the left ventricle to force at least a large portion of the air through the capillaries into the veins. The primary effect of embolism of the vessels of the brain is to produce acute cerebral ischæmia, the intensity of which depends upon the number and size of the vessels which have become obstructed by the air-emboli. This anæmia soon gives rise to intense engorgement of the collateral vessels, and the vessels behind the point of obstruction, an engorgement to such an extent, that it often leads to rupture of capillary vessels and hemorrhage into the paravascular tissues. Unless the amount of air injected is sufficient to prove rapidly fatal by causing suspension of functions of the cerebro-spinal centres, the animal dies of asphyxia from embolism of the pulmonary artery, the same as though the air had been injected directly into the veins. The left ventricle, from its greater thickness and consequently more powerful contractions (as compared with the right ventricle), is better adapted to overcome the increased resistance, and hence the air is rapidly forced through the systemic capillary circulation into the veins and right side of

the heart. On this account an equal amount of air injected into arteries is not so dangerous as when introduced directly into veins, as a certain percentage of it never reaches the veins and right side of the heart. Arterial air-embolism is attended by an additional source of danger, which consists in the greater tendency to coagulation of blood in the heart and vessels, as was noted in several experiments.

XIII. *Direct Intra-Cardiac Insufflation of Air.*

These experiments were made with a view of demonstrating by ocular inspection, that sudden over-distension of the cardiac muscles will arrest their contractility and will thus produce paralysis of the heart in the diastole.

Experiment No. 1.—Medium sized cat. After the animal was fully under the influence of ether, respirations and heart's action being normal, the chest was rapidly opened and the heart exposed. Its movements remained regular. From the sudden ingress of air into the cavity of the chest, the left lung collapsed, and it was found necessary, in order to prevent asphyxia, to make artificial respirations on the right side of the chest. The pericardium having been opened, the right ventricle was punctured with the needle of an aspirator, and air injected in sufficient quantity to distend the right chambers of the heart, which immediately ceased to contract. On emptying the right ventricle by withdrawing the air and blood, the rhythmic movements of the organ were restored. These manipulations were repeated at least half a dozen times, and always with the same uniform and constant results. Half an hour after opening the chest all movements of the heart ceased. The heart was now removed and immersed in water at a temperature of 105° F., when it again commenced to pulsate at the rate of 120 beats per minute. After the lapse of about two minutes it was removed from the warm water, still pulsating, and transferred to cold water, when all movements ceased instantaneously, and all efforts to revive them proved futile.

Experiment No. 2.—Large Maltese cat. Heart exposed, right ventricle punctured, and 11 c. cm. of air injected with the result of

immediately arresting the pulsations of the right ventricle. When the air was withdrawn, the regular movements were renewed. This experiment was repeated on both ventricles eight or ten times in the course of half an hour with the same results. The auricles during all this time continued to contract. Ten minutes after the last puncture and evacuation the ventricles were still pulsating.

Remarks.—Both of these observations support the statement that sudden over-distension of any of the cavities of the heart by inflation with air, will arrest its movements in the diastole, and that upon the removal of the increased intra-cardiac pressure, the movements of the organ are restored. It was found that by continuing the injecting force after the right ventricle had become distended, air could be forced readily through the pulmonary capillaries into the left side of the heart. In some instances one of the cavities of the heart was kept over-distended and at rest for at least a minute, and yet on removing its contents the rhythmic movements were resumed.

XIV. *Aspiration of Right Ventricle for Air-Embolism.*

Being satisfied of the fact that sudden over-distension of the right ventricle may and does produce paralysis in the diastole, it appeared reasonable to resort to some direct measure in removing the cause of over-distension in grave cases of venous air-embolism. Puncturing and aspiration of the right ventricle seemed to offer the best chances for accomplishing this object. In the following experiments artificial venous air-embolism was produced in the usual way, when the right ventricle was punctured with the needle of an aspirator two millimetres in diameter, and the contents of the right side of the heart removed by aspiration. The region over the heart was shaved and disinfected, and the exact point of puncture located before the animal was anæsthetized. The needle was always carefully disinfected by passing it through the flame of an alcohol lamp, and immersion in carbolized water. When the puncture was made, the needle was advanced first only sufficiently deep to bury the opening in its point beneath the tissues, when a vacuum was created

in the aspirator so that the entrance of the needle into the ventricle would be promptly announced by the escape of spumous blood. By following this precaution, additional injury to the heart, by pushing the needle further into the cavity of the ventricle, was avoided, and at the same time a prompt escape of blood was secured. Experience taught me that it was very important not to push the needle too deeply into the cavity of the ventricle; not only for fear of inflicting additional injury to the endocardial lining opposite the point of entrance, but more particularly with a view of removing the free air which would naturally occupy the highest point in the cavity, if it was not intimately mixed with the blood. The needle was always directed obliquely from below upwards for the twofold purpose of making a valvular wound in the ventricle and of avoiding unnecessary injury to the endocardium.

Experiment No. 1.—Dog, weight 31 pounds. 60 c. cm. of air were injected into the left jugular vein. The animal was seized immediately with a convulsion, followed by collapse. Heart's action tumultuous. Churning sounds over heart were distinctly perceptible. Respirations rapid and superficial. Right ventricle was punctured, and 120 c. cm. of spumous blood were withdrawn. Pulsations of heart became more feeble, and as respirations had ceased and could not be restored, the chest was opened. The right ventricle was found moderately dilated and still contracting. Slight contractions of lower portion of left auricle. Left ventricle contracted, but without motion. The needle of the aspirator was then introduced into the right auricle, which was distended and tympanitic. After emptying it, it commenced to contract. All contractions ceased half an hour after the air was introduced. Faradic currents had no effect upon the heart after it had ceased to contract. The pericardium contained air in considerable quantity and about four c. cm. of fluid blood. Air was found in the right side of the heart, the left ventricle, coronary veins, and in the large veins in the immediate vicinity of the heart. Puncture about an inch from septum, and nearly equidistant between the apex and base of heart. No injury of endocardium on opposite side.

Experiment No. 2.—Sheep, weight 95 pounds. Before operation respirations 86, pulse 140. Injected 150 c. cm. of air into left

jugular vein, which was immediately followed by convulsions and involuntary discharges. Aspiration of right ventricle and removal of 150 c. cm. of spumous blood. As the animal still presented an asphyxiated appearance, the ligature was removed from the distal end of the jugular vein, and about 120 c. cm. of blood were allowed to escape. The animal showed no signs of improvement, and died five minutes after the injection was made, having made only five attempts at respiration during the time. Pericardium contained a small coagulum and a few bubbles of air. Right ventricle, distended and tympanitic, contained spumous blood. Two punctures were found over the middle of the right ventricle about one-half cm. apart; the second was made during the convulsive movements of the animal, which necessitated a partial withdrawal and reintroduction of the needle. On the inner surface each puncture was hermetically sealed by a minute thrombus which projected only very slightly into the cavity of the ventricle. No injury of opposite wall of ventricle. In the left ventricle, which was firmly contracted and empty, a small filiform coagulum projected through the aortic orifice into the aorta.

Experiment No. 3.—Old dog, weight 40 pounds. Injected 45 c. cm. of air into left jugular vein. Churning sounds over heart loud and distinct. Heart's action labored; respirations exceedingly rapid. No convulsion, but profound coma and involuntary discharges. A little more than one minute elapsed between the introduction of air and aspiration, during which time the heart's action became more and more embarrassed, and all of the symptoms became so grave, that death seemed unavoidable. But as the needle reached the heart, a slight impulse was imparted to it by the feeble pulsations, and 120 c. cm. of air and spumous blood were removed. The heart's action improved immediately, but the dyspnœa, although less severe, was still quite prominent. Consequently an additional 120 c. cm. of blood were allowed to flow from the peripheral extremity of the jugular vein, which promptly relieved the urgent symptoms. Ten minutes after the injection, respirations were 240 per minute; four minutes later 360, and after eight minutes more 120. After forty minutes the stupor disappeared and the animal was able to walk. One hour after the operation, respirations were 20 and pulse 160 per minute. At this time the animal

walked home—a distance of nearly two miles—and subsequently suffered no inconvenience from the operation.

Experiment No. 4.—Dog, weight 75 pounds. Injected into left jugular vein 90 c. cm. of air, which produced intense difficulty in breathing and profound stupor. Sensation and reflex movements suspended. Heart's action exceedingly feeble and rapid. Churning sounds distinct. The aspirator and needle were kept in readiness to be used promptly on the approach of symptoms indicative of impending death. Two minutes after the injection the animal was apparently in a dying condition, when the needle was plunged into the right ventricle, and from its rapid oscillations it was apparent that the heart's action had not entirely ceased, but was very rapid and feeble. As quickly as possible 180 c. cm. of spumous blood were withdrawn, which promptly relieved the most urgent symptoms; but as the respirations still remained rapid and labored, the ligature was removed from the distal end of the jugular vein, and 240 c. cm. of blood were allowed to escape. Eight minutes later after the injection, the churning sounds over the heart were still present, but faint. Respirations 144; pulse 130. When the animal had recovered from the immediate effects of the operation, it was noticed that the posterior extremities could be moved only with difficulty, the gait being slow and staggering. For twelve hours the animal was dull and stupid, remaining in the same place without change of position. The drowsiness continued for thirty-six hours, after which the animal became lively, and went on to complete recovery without any further interruption. In this case it was apparent that the animal would have died from the large volume of air which had been injected, but for timely use of the needle and aspirator. Three weeks after the experiment the animal, being in perfect health, was killed by an arterial injection of air. No signs of pericarditis, endocarditis, or myocarditis. Puncture in ventricle indicated by a minute faint cicatrix.

Experiment No. 5.—Old dog, weight 25 pounds. In this case the aspirator was used a few seconds after the insufflation of air was made, and the result shows how important it is to withdraw the air from the ventricle before it has had time to escape into the pulmonary artery. Immediately after the injection of 90 c. cm. of air into the left jugular vein, the animal became comatose. Respiration

exceedingly rapid and heart's action imperceptible. The right ventricle was punctured at once and as soon as a part of its contents had been removed the impulse of the heart was imparted to the needle which could be distinctly seen and felt. Spumous blood and air to the amount of 150 c. cm. were withdrawn. The dog rallied rapidly after the aspiration, being able to walk in less than half an hour. He manifested no pain or discomfort from the air-embolism or operation. The volume of air in this case was three times as large as in the previous experiment, when the weight of the animals is compared. The immediate effects of the air were expended upon the heart as was made evident by the temporary cessation of its pulsations, and yet death was prevented by the prompt removal of the excessive intra-cardiac pressure. The recovery was more rapid than in the previous cases, from the fact that not so much air had passed into the pulmonary artery and more was removed by aspiration. Two weeks later, the animal being in perfect health, 60 c. cm. of air were injected into the right jugular vein, which produced death in a few minutes, showing conclusively that the recovery after the first insufflation was due to the aspiration. Cicatrix of puncture plainly visible. No signs of inflammation in the pericardium, endocardium, or substance of the heart.

Experiment No. 6.—Young dog, weight 28 pounds. In this experiment 120 c. cm. of air were thrown into the left jugular vein in divided doses of 30 c. cm. each in rapid succession. During the third injection the animal howled and became comatose, and immediately after the last injection the heart's action ceased and respirations were reduced to a few irregular gasps. The right ventricle was punctured and 120 c. cm. of spumous blood and air were withdrawn, without producing any effect upon the heart. Artificial respiration was resorted to without any better result. The chest was opened at once. The pericardium contained 4 c. cm. of dark, venous blood and a few air-bubbles. Right auricle continued to contract forty-eight times per minute. Left auricle and ventricle were distended and contained spumous blood. The needle had punctured the right auricle instead of the ventricle. All muscles, voluntary and involuntary, reacted promptly to the faradic current, but it had not the slightest effect upon the heart.

Experiment No. 7.—Young dog, weight 10 pounds. Injected 45 c. cm. of air into the left jugular vein. At the end of injection

the dog howled, and the heart's action ceased almost instantaneously. The right ventricle was aspirated and 60 c. cm. of spumous blood were withdrawn. Artificial respiration was performed and the faradic current was applied, when the dog made a few ineffectual attempts at respiration before he died. On examination the pericardium was found to contain a slight amount of dark fluid blood and a few air-bubbles. Both auricles contracted about forty-eight times in a minute. Right ventricle arrested in diastole. Puncture was found near base of ventricle. Right ventricle was again aspirated, and the faradic current was applied directly to the heart and the needle, without producing the slightest effect. Right ventricle and pulmonary artery contained spumous blood. Left ventricle contracted and nearly empty.

Experiment No. 8.—Adult dog, weight 24 pounds. Before operation, respirations 80; pulse 100 in a minute. Injection of 30 c. cm. of air into left jugular vein. Immediately after injection great restlessness, dyspnœa, and tumultuous action of heart. Churning sounds loud and distinct. Removed 90 c. cm. of spumous blood from right ventricle by aspiration. After the aspiration the improvement in respiration and pulse was marked. Recovery complete, and not attended by any complications.

Experiment No. 9.—Same animal as in experiment No. 3. Animal in perfect health; time since first operation three weeks. Injection of 40 c. cm. of air into right jugular vein. Symptoms the same as before. At the end of three minutes and a half the pulsations of the heart ceased, and the respirations were nearly suspended. The right ventricle was now punctured and 250 c. cm. of spumous blood were removed. No motion imparted to needle. Faradization and artificial respiration were ineffectual in restoring either the heart's action or respiration. On examination 30 c. cm. of dark fluid blood were found in the pericardium. No evidences of inflammation from former puncture. Right ventricle moderately distended with a clot of spumous blood. Point of second puncture one-half inch from coronary artery. The location of first puncture was marked near the base of the right ventricle by a faint minute cicatrix upon the pericardial surface, while the endocardium at a corresponding point showed an old circumscribed spot of ecchymosis, but no evidences of inflammation. Left ventricle contained no air and

only a minute quantity of fluid blood. Both auricles nearly empty. Pulmonary artery contained air and spumous blood. This experiment demonstrates positively and conclusively the value of early aspiration of the right ventricle in venous air-embolism, where death is threatened by over-distension of the right side of the heart, or by asphyxia. In the former experiment a larger amount of air was injected, but the animal was saved by an early aspiration by which a large amount of air was removed before it had time to accumulate in the pulmonary artery. It also illustrates the utter uselessness of resorting to any kind of mechanical interference after a fatal dose of air has once passed beyond the semilunar valves into the pulmonary artery. In such instances the air has passed beyond our reach, and will inevitably lead to a fatal result by asphyxia.

Experiment No. 10.—Adult dog, weight 18 pounds. Injection of 60 c. cm. of air into left jugular vein. Immediate collapse; churning sounds distinct. Aspiration of 100 c. cm. of air and spumous blood from right ventricle. Three minutes after the insufflation, respiration, and heart's action had ceased. No air or blood found in the pericardium. Needle puncture equidistant between base and apex of right ventricle, and about two cm. from septum. Right auricle contracting feebly forty times a minute. Right ventricle dilated, fibrillary contractions at apex only; right ventricle and pulmonary artery contained spumous blood. Left auricle and ventricle contained only a slight amount of fluid blood and a few air bubbles.

Remarks.—These experiments serve to illustrate the following interesting points in pathology and surgery: 1. The heart can be punctured with a perfectly aseptic, medium-sized needle of an aspirator, without any great immediate or remote danger. 2. Aspiration of the right ventricle for venous air-embolism, when done early enough (before a fatal dose of air has been forced into the pulmonary artery), must be considered in the light of a life-saving operation.

Although the needle used in all of these experiments was two millimetres in diameter, the hemorrhage into the pericardium was never sufficient in quantity to prove a source of danger. The largest quantity found was 30 c. cm. (in experiment No. 9).

In a number of cases no trace of blood was found in the pericardium. Against my expectations the same observations were made in regard to air. In one case the pericardium contained about 60 c. cm. of air, while in most cases only a few isolated air-bubbles were seen, while in others no trace of it could be found. In all cases where the animal survived the operation and was killed from a few days to three weeks subsequently, no trace of inflammation could be found either in serous membranes, muscular tissue, or adjacent organs. Even adhesion between the parietal and visceral layers of the pericardium at the point of puncture never occurred. The point of puncture was usually marked by a faint minute cicatrix in the visceral pericardium, unaccompanied by any pathological changes in the substance of the heart. We must take it for granted that if effusion of blood or air occurred into the pericardium in the animals which recovered, these adventitious substances caused no irritation, but were promptly removed by absorption.

With the exception of the subject of experiment No. 8, it may be safely assumed, that all of the animals would have died had aspiration not been performed, so that the operation saved at least three of the animals out of ten, or the whole number of experiments. The most conclusive proof of this statement was furnished by the subjects of experiments Nos. 3 and 5, as these animals died during subsequent experiments from the introduction of a much smaller quantity of air. The question naturally arises, Why were not all of the animals saved by the aspiration? In reply it may be stated that the amount of air injected in most instances was large, more than was necessary to produce a fatal result. Another element of failure consisted in the postponement of the aspiration until a fatal dose of air had passed beyond the reach of the aspirator. To prevent death by air-embolism it is essential to remove the air from the right ventricle as soon as possible after its entrance, before a fatal embolism of the pulmonary artery has had time to take place. In some of the experiments a fatal result might have been probably prevented by removing a larger quantity of air and blood with

the aspirator, as in some instances the condition of the animal was improved by a subsequent venesection from the jugular vein.

XV. *Catheterization and Aspiration of Right Auricle for Venous Air-Embolism.*

In grave cases of accidental air-embolism it would be a desideratum to be in possession of some means, by which the air could be removed directly from the right side of the heart in the shortest possible space of time and with the simplest instrument. An aspirator is not always at hand, and less frequently in a proper condition to be used on such short notice. It appeared to me that inasmuch as the accidental introduction of air usually takes place through wounds in one of the jugular veins, a catheter might be introduced through the wound into the vein and from there passed directly into the heart, and through it the air and spumous blood could be withdrawn by aspiration. A catheter is almost always at hand, and its introduction would require only a few moments of time. The following experiments were made to test the feasibility of this procedure.

Experiment No. 1.—Adult dog, weight 24 pounds. The right jugular vein was opened and 60 c. cm. of air were introduced in the usual way. The breathing became very difficult and the heart's action labored. The animal was comatose, pupils dilated. As soon as it could be done, a number six, English scale, gum elastic catheter, which had previously been made aseptic, was introduced into the wound, and passed into the heart—a distance of 15 cm.—and with an exhausting syringe 250 c. cm. of air and spumous blood were removed. The animal recovered rapidly from the immediate effects of the air-embolism and operation, and subsequently manifested no symptoms of disease.

Experiment No. 2.—Young dog, weight 30 pounds. Injected 100 c. cm. of air into left jugular vein. The heart's action ceased almost instantaneously. The same catheter was introduced and pushed forward a distance of 18 cm. and about 120 c. cm. of air and spumous blood were withdrawn by the mouth. At this time

respiration ceased, and as no pulsations of the heart could be felt, the chest was opened at once. Fibrillary contractions of right ventricle which was distended and tympanitic. Left auricle and ventricle contracted and motionless. Right auricle contracted regularly 80 times a minute. Faradic and galvanic currents had no effect on ventricles. Right auricle responded to a galvanic current slowly interrupted. The catheter was again introduced, and its course observed as it entered the right auricle. It was found impossible by any manipulation to pass it from the auricle into the ventricle. In every instance it passed from the right auricle directly into the inferior vena cava. The right side of the heart was filled with water to test the possibility of removing fluids from the right auricle without introduction of the catheter into the chamber. A catheter with an open extremity was used. No amount of suction force succeeded in removing any of the fluid, until the open end of the catheter reached the auricle, when both chambers could be readily emptied. As long as the end of the catheter remained in the vein, the walls of the vein in front of the catheter would invariably collapse and close the opening in the catheter completely on applying suction force. This satisfied me that in order to remove air or blood from the right side of the heart, it is necessary to introduce the catheter as far as the auricle. It was also evident that in case the catheter be introduced too far, its distal end would pass into the inferior vena cava, and on aspiration only venous blood would be withdrawn, not air and spumous blood from the right chambers of the heart, for the removal of which the procedure was intended. On opening the right side of the heart, a large spongy clot of spumous blood was found in the right auricle, which extended for some distance into the ventricle. Left ventricle contained only a small amount of fluid blood and a few air-bubbles. As the heart's action had ceased before catheterization, death was undoubtedly due to the insufflation of air and not to the formation of a thrombus in the heart.

Experiment No. 3.—Adult dog, weight 20 pounds. Injected 30 c. cm. of air into right jugular vein. Catheterization and aspiration of right auricle, by which 90 c. cm. of air and spumous blood were removed when catheter became impermeable by a thrombus in its interior and was removed. Before emptying the right side of the heart the animal was comatose, breathing and heart's action exceedingly rapid. After aspiration rapid improvement and complete re-

covery. If the catheter in this instance had been permitted to remain longer in the auricle, the thrombus which formed in the interior of the instrument would undoubtedly have increased rapidly, and would have extended to the auricle, in which event death from thrombosis would have been unavoidable.

Experiment No 4.—Old dog, weight 40 pounds. Before operation respirations 16; pulse 100 per minute. Injection of 90 c. cm. of air into left jugular vein. Passed a No. 5 Nélaton's catheter into right auricle, a distance of 27 cm., and removed 250 c. cm. of air and spumous blood. Immediately after the insufflation the animal uttered a howl and became comatose. Pulse 250 per minute, respirations so rapid that they could not be counted. Seven minutes after catheterization heart's action very irregular, tumultuous, and about 300 beats per minute. Two minutes later respiration ceased, after which the heart's action became slow and feeble, only to cease a few seconds later. Internal mammary and coronary veins filled with air. Left side of heart contained a small amount of bright red, frothy blood. Right ventricle moderately dilated, showed fibrillary contractions. Superior and inferior vena cava, right auricle and ventricle contained a continuous, soft thrombus, the oldest portion of which corresponded to the superior vena cava. The upper portion of the clot in the inferior vena cava contained air. Death in this instance was due to the formation of a thrombus.

Experiment No 5.—Adult dog, weight 25 pounds. Injection of 60 c. cm. of air into left jugular vein produced immediate collapse, distressing dyspnœa, and great rapidity and irregularity of heart's action. The dog howled when the heart's action ceased, and respiration became gasping. Some delay was experienced in the introduction of the catheter, and when the instrument was in place all signs of life had disappeared. About 50 c. cm. of spumous blood were withdrawn without any signs of improvement. Artificial respiration and faradization were resorted to without producing the slightest impression. On opening the chest it was observed that the right auricle contracted about 30 times a minute. Irregular fibrillary contractions of apex of right ventricle. Right side of heart moderately distended with spumous blood. No thrombus. Left ventricle almost empty.

Experiment No. 6.—Adult dog, weight 15 pounds. Injection of 60 c. cm. of air into right jugular vein. In this case the catheter was connected with a rubber-tube three feet in length, which was kept under water; then the instrument was pushed along the jugular vein into the right auricle. The injection of air was followed by the most urgent and distressing symptoms. Only a small amount of dark venous blood escaped through the rubber-tube until the catheter reached the right auricle, when a gush of spumous blood escaped from the tube under water. As the blood did not continue to escape, about 60 c. cm. of spumous blood were withdrawn. After the aspiration the symptoms improved rapidly, and within twenty-five minutes, with the exception of a slight excess in the respiratory movements, the animal appeared to be perfectly well. The ultimate recovery was complete and not disturbed by any complications.

Experiment No. 7.—Adult dog, weight 48 pounds. Before operation pulse 150; respirations 28. Injection of 120 c. cm. of air into right jugular vein. The animal howled and collapsed. Respirations increased at once to 100 per minute. Heart's action tumultuous, and churning sounds loud and distinct. Passed a Nélaton's catheter into the right auricle, and aspirated 360 c. cm. of air and spumous blood. No signs of improvement followed, and in fifteen minutes the animal was dead. At the post-mortem examination the internal mammary veins were found distended with air. Right side of heart tympanitic and distended. Slight fibrillary contractions of right auricle. Apex of right ventricle contracted regularly 22 times in a minute. Coronary veins filled with air. A thrombus had formed, which extended from the superior vena cava into right auricle, ventricle, and for a distance of 25 cm. into the inferior vena cava. Pulmonary artery contained no thrombus, but was distended with air and spumous blood. Left ventricle contracted, contained a small amount of fluid blood and air-bubbles. It was quite evident that the thrombus had primarily formed around the catheter, and by new additions it finally reached the distal portion of the inferior vena cava. Death was produced by thrombosis.

Remarks.—The therapeutical value of catheterization and aspiration of the right auricle for air-embolism is made apparent by the following tabular arrangement of the experiments:—

No. of experiment.	Weight of animal.	Amount of air injected.	Amount of air and blood removed.	Result.	Cause of death.
1	24 pounds.	60 c. cm.	250 c. cm.	Recovery.	
2	30 "	100 "	120 "	Death	Thrombosis.
3	20 "	30 "	90 "	Recovery.	
4	40 "	90 "	250 "	Death	Thrombosis.
5	25 "	60 "	50 "	"	Air-embolism.
6	15 "	60 "	60 "	Recovery.	
7	48 "	120 "	360 "	Death	Thrombosis.

From this table it will be seen that of seven animals subjected to the operation, three recovered and four died. The amount of air injected in the subjects of experiments Nos. 1 and 6 was sufficient to destroy life, so that we can safely assume that the animals would have died, but for the speedy resort to catheterization and aspiration. In the subject of experiment No. 3 recovery might have taken place without the operation, as the relative quantity of air was much smaller. In the four fatal cases death was produced only once from the presence of air, while the direct cause of death in the remaining three was due to thrombosis. All possible precautions were exercised to prevent the accident. The catheter was made aseptic by thorough cleansing and immersion in a five per cent. solution of carbolic acid. Before the operation the instrument was kept ready for use in an alkaline solution of the temperature of the body, and yet this accident happened in three out of seven cases. It is now asserted that the introduction of aseptic bodies into the circulation does not give rise to thrombosis, and it may be possible that some of the instruments used, in spite of the pains taken to render them aseptic, were still not in a fit condition to be used for such a purpose. Catheterization and aspiration of the right auricle for air-embolism compare favorably with puncture and aspiration of the right ventricle as a life-saving procedure, but the former operation is more dangerous on account of the tendency to the formation of a thrombus within or around the catheter. If this formation of thrombus could be avoided with certainty, catheterization and aspiration of the right auricle

would recommend itself as the most expedient and reliable therapeutic agent in cases where life is threatened by air-embolism.

XVI. *Prophylactic Treatment of Air-Embolism.*

As clinical experience and experimental research have shown that the admission of air into veins is not an infrequent occurrence and often has resulted in death, it is the duty of the surgeon in extirpating tumors, which are in close proximity to the large veins within the area of the "danger zone" to resort to measures which will prevent the ingress of air in case a vein is accidentally wounded. The following precautionary means deserve consideration: 1. Position. 2. Compression. 3. Ligature. 4. Aseptic tampon.

1. *Position.*—Regular respiratory movements of the chest are necessary to maintain the equilibrium between the arterial and venous circulation, and should always be secured in operating in close proximity to veins at the base of the neck, as during a sudden deep inspiration the direct aspiratory effect of respiration in the veins extends to some distance, constituting thus the direct cause of entrance of air into an open vein. Before anæsthetics were used, Poiseuille advised that the patient should be instructed not to make any deep inspiratory movements during the operation. At the present time the same object is better obtained by keeping the patient thoroughly and continuously under the influence of the anæsthetic. Gerdy aimed to prevent the aspiratory effect of respiration by recommending compression of the thorax, but the advice is more useful in theory than in practice, as suspension of the thoracic movements for any length of time would necessarily interfere with respiration and cause death by asphyxia.

The venous circulation is greatly influenced by position. In the elevated position the blood gravitates towards the heart and the veins are emptied. If in this position a vein is wounded, and the walls of the vessel do not collapse on account of some anatomical peculiarities, or from rigidity of the vein wall itself,

due to pathological changes, a vacuum is formed and air enters. The entrance of air into a vein distended with blood is a physical impossibility. In reading the clinical histories of cases where air entered veins during operations, we are met by the fact that in almost all of the cases, the wounding of the vein was not followed by any considerable loss of blood, and we are usually told that the air entered almost immediately after the vessel was injured, which would indicate that the vein was empty or nearly so at the time it was wounded. In a number of cases the bloodless condition of the vein attracted the attention of the operator, and is particularly emphasized in the description of the cases. As long as the interior of a vein and the lumen of a wound in its walls are occupied by a continuous stream of blood there is no danger that air will be admitted. Statistics tend to show that the accidental admission of air into veins was more frequent before anæsthetics were used, a fact which we can only explain by assuming that the patients were then usually placed in a sitting or semi-recumbent position during the operation, positions favorable to the return of venous blood from the cervical region. On the other hand, the safe administration of an anæsthetic necessitates the horizontal position in which the veins of the neck become distended with blood. In the former instance the return of venous blood in the vessels of the upper portion of the body was favored by the elevated position, and the vacuity thus produced in the veins constituted the most important and potent factor in determining the entrance of air. In the horizontal position, the veins of the neck are never empty, and to effect entrance of air the exciting cause must operate with increased intensity. On this account the horizontal or dependent position of the region of the body to be operated upon, recommends itself as the simplest prophylactic measure against the accidental entrance of air into wounded veins. This position increases the risk of hemorrhage from the injured vein, but this accident in its immediate effects is less disastrous to the patient and more readily under the control of the surgeon than air-embolism.

2. *Compression.*—Compression of a vein between the seat of

operation and the heart for the purpose of guarding against the entrance of air may be divided into intermediate and direct. Intermediate compression is only applicable in cases where the external jugular vein is in danger of being wounded. If the tumor to be extirpated is not located too low in the region of the neck uninterrupted digital compression of the external jugular vein just above the clavicle can be relied upon in preventing entrance of air through this vessel. The internal jugular and axillary veins are so deeply situated that intermediate digital compression cannot be relied upon in the prevention of air-embolism, consequently it becomes necessary to resort to direct compression whenever it becomes necessary to guard against this accident. Warren recommended that in the extirpation of tumors which are in close proximity to the large veins of the neck and axilla, the separation of the pedicle should be reserved until the last in order to enable the operator to compress the vein between the seat of operation and the heart more effectually, should the vessel be injured during the operation.

Langenbeck advised that the vein on the distal and proximal sides of the tumor should be exposed to sight as a preliminary measure before attempting the removal of the tumor with a view to facilitate direct compression in case the vessel should be opened during the dissection. The adoption of either of these precautions would successfully prevent a second ingress of air, but would not protect the patient against the dangers arising from the first dose. Permanent central compression augments the danger of wounding the vein from the turgidity of the vessel which it produces, while distal compression would be attended by the same risk, only in an opposite way, by rendering the vessel empty and consequently more difficult of recognition. If the attachments of the tumor to the vein are of such a nature that they can be separated without great danger of injuring the vessel, then the vein should be isolated on the proximal side and the vessel compressed with a hæmostatic forceps which will afford greater safety than digital compression, at the same time the field of operation is not obscured and narrowed by the

fingers and hand of the assistant, an item of great importance in operating in the regions of the neck or axillary space.

Injury of the vein in such instances will be promptly announced by hemorrhage from the distal portion, but the entrance of air into the right ventricle is made impossible by the prophylactic compression of the vessel between the seat of operation and the heart. Should the vein be wounded, any further ill effects arising from this accident can be prevented by applying a lateral ligature or tying both ends of the vein according to the size of the wound, before removing the compressing forceps. In all operations where any of the large veins are in danger of being wounded the position of the parts should be such as to retain as nearly as possible their normal anatomical relations. The operator should make haste slowly, and identify every structure before using cutting instruments. In extirpating tumors that are deeply situated, the external incision should always be large so as to afford free access to the base or deepest portion of the growth to be removed. A pair of Billroth's retractors will do good service during the deep dissection.

Hemorrhage must be carefully arrested as it occurs, in order to enable the surgeon to see what he is doing. Isolation of the vein from the tumor must be accomplished by the use of blunt instruments, and all firmer attachments should be carefully examined and identified before using the knife or scissors. As it is usually impossible, or, at any rate, impracticable to find the vein in the tumor or its immediate vicinity, the rule should always be followed to expose the vessel for some distance from the tumor on the proximate side, and then follow it by carefully separating the attached tissues by means of blunt instruments. Under antiseptic precautions the jugular vein can be isolated for a great distance without fear of compromising the vitality of its tissues or causing thrombosis. When a tumor is attached to the jugular vein it is a dangerous practice to make traction on the tumor for the purpose of facilitating the deep dissection, as this procedure will disturb the normal anatomical relations of the vessel and thus expose it to greater risk of being wounded. Free access to the base of the tumor must be secured without

displacing the subjacent vein. As soon as the tumor has been isolated from the surrounding tissues a thorough examination should be made of the extent of its base and its relations to the subjacent vein before proceeding any further with the dissection. If the conclusion is reached that the vein is so intimately connected with the base of the tumor that it is in great danger of being injured during the further progress of the extirpation, it becomes necessary to resort to direct compression of the distal and proximal portion of the vein. By isolating and compressing the vein on each side of the tumor both excessive turgidity and emptiness of the intervening portion of the vein are avoided. The amount of blood in the vein between the two points of compression can be regulated, and only a sufficient quantity is allowed to remain to indicate the exact location of the vessel. Compression is again made with hæmostatic forceps. After the complete removal of the tumor the forceps on the distal side of the vein is removed first, in order to test the integrity of the vessel and with a view to prevent any possibility of the entrance of air. If no hemorrhage takes place we may be satisfied that the vein has not been injured, and the remaining pair of forceps can be safely removed. In such cases the exposed portion of the vein should always be covered with the adjacent tissues by deep sutures *en étages* of catgut before closing the wound. If the vein has been wounded a lateral ligature is applied, or both ends are ligated after complete division of the vessel before the forceps between the vein wound and the heart is removed. By following this plan we accomplish the following objects: 1. Hemorrhage from the injured vein is slight and perfectly under the control of the surgeon. 2. Absolute security against the entrance of air. 3. A more thorough removal of the tumor.

3. *Ligature.*—In all cases where a large vein passes through the substance of the tumor and where isolation of the vessel appears inexpedient or impossible, double ligation recommends itself as the safest prophylactic measure both against hemorrhage and air-embolism. As soon as the base of the tumor is reached the vein is exposed on each side and tied with catgut, and the

intervening portion is extirpated with the tumor. By adopting this plan we secure for the patient absolute protection against hemorrhage and air-embolism. Adequate collateral circulation is established in a short time, so that ligation or excision of the internal jugular or axillary veins can be done under antiseptic precautions without danger of causing serious disturbance of the cerebral circulation in the former, or gangrene of the arm in the latter instance. Isolation and ligation of the vein on the proximal side of the tumor should always be resorted to, when injury to the vein is unavoidable. In case the prophylactic ligation of the vein on the distal side of the tumor is rendered difficult or impossible by existing circumstances, the following course can be pursued. After ligation of the vein on the proximal side of the tumor a hæmostatic forceps is applied a little distance from the ligature on the side of the tumor, and the vessel is divided between the forceps and the ligature. The ligature prevents the entrance of air, and the forceps serves the purpose of a temporary hæmostatic agent during the extirpation of the tumor. The base of the tumor with the vein is detached when the vessel on the distal side of the tumor is sought for and tied, and the remaining attachments of the tumor, including the vein, are cut. This plan affords protection against the entrance of air, but during the separation of the tumor hemorrhage may occur from accidental wounds of the distal portion of the vein, which, however, can be readily controlled by compression with hæmostatic forceps until the vessel is permanently secured on the distal side of the tumor.

4. *Aseptic Tampon.*—If a large vein has been wounded which from its location is inaccessible to the ligature, permanent compression with a graduated aseptic tampon will arrest the hemorrhage and prevent further ingress of air.

XVII. *Operative Treatment of Air-Embolism.*

Considering the infrequency of air-embolism as compared with the number of vein wounds, it is not surprising to find that, as a rule, surgical writers have little, if anything, to say on the

subject of the prevention and treatment of air-embolism. It must be apparent to all, that in following the rules laid down when we considered the prophylactic treatment, the accidental introduction of air into veins is prevented almost to a certainty, and at the same time they will also furnish a safe guide in the prevention and management of venous hemorrhage when operating within the area of the "danger zone." If the preventive treatment is carefully carried out, air-embolism will become one of the rarest accidents in surgery. As even with the greatest care an accident of this kind might happen, it becomes necessary to allude to the operative treatment of air-embolism. The occurrence of the accident is sometimes announced by an audible hissing or sucking sound, and is always followed almost instantly by a well-marked train of distressing symptoms which are directly referable to a mechanical obstruction to the circulation in the right side of the heart and the pulmonary artery. The treatment must depend on the quantity of air which has entered, and the severity of the symptoms produced by it. The therapeutic measures should be aimed towards meeting the following indications: 1. Prevention of further ingress of air. 2. Administration of cardiac stimulants to sustain the action of the heart. 3. Venesection to relieve intravenous pressure. 4. Aspiration of air from the right side of the heart to prevent over-distension and paralysis of right ventricle.

1. When air has entered through a gaping wound in a vein during inspiration there is great danger that the same occurrence will repeat itself during successive respiratory movements of the chest, hence the first object of treatment consists in closing the wound in the vein. This is most quickly done by digital compression, which is continued until the vein has been rendered impermeable by ligature or permanent compression. If the symptoms are urgent no time should be lost in securing the vessel until the patient has rallied from the immediate effects of the air-embolism. It is also necessary to postpone the permanent closure of the vein until the time has elapsed for any indications to arise which would call for venesection or operative removal of the air from the right side of the heart, as in such

cases it may become necessary to utilize the wounded vein as a route for the introduction of a catheter into the heart. When the time has arrived for closing the vein the finger should not be removed suddenly from the vessel for fear of causing a repetition of the accident; it should remain *in situ* until the vessel can be compressed on the proximal side of the vein wound by an assistant. The exact location of the wound will now be probably indicated by escape of blood from the distal end of the vein when the vessel can be seized with a forceps, and after its isolation, a double ligature is applied. If it is found impossible to ligate the vein, then the vessel is compressed in the wound by means of a graduated aseptic tampon retained in place by uninterrupted pressure until definitive closure of the wound has taken place.

2. The heart's action must be supported by position and by the use of cardiac stimulants, which are best administered subcutaneously and by inhalation. The patient must be placed in the horizontal position with a view of guarding as much as possible against the occurrence of cerebral anæmia as well as to lessen the intravascular pressure to a minimum. If the quantity of air admitted is not sufficient to produce instant death by paralysis of the right side of the heart in the diastole, the symptoms which follow always point towards an embarrassment of the circulation in the right side of the heart and the pulmonary artery, which are always accompanied by dyspnœa, combined with evidences indicating the existence of acute cerebral ischæmia. If death does not take place within a few minutes from obstruction to the circulation through the pulmonary artery by the presence of air in that vessel, then the contractions of the right ventricle will force at least a part of the air through the pulmonary capillaries into the general circulation, thus preventing death by asphyxia. As soon as the air-embolism of the pulmonary artery has been relieved the most urgent symptoms subside, as the air-emboli in the arterial system are distributed over a larger area and consequently produce an embolism of lesser extent and gravity in more distant and less essential organs. The safety of the patient depends on the capacity of

the right ventricle to force the air through the pulmonary into the general circulation, in other words, upon the time which is required in removing the emboli from the pulmonary artery into the systemic circulation.

Nitrite of amyl, from its stimulating properties upon the heart and from the rapidity of its action, would recommend itself in the form of inhalation as the most efficient drug in preventing threatened syncope. The temporary dilatation of the small bloodvessels would tend to produce, at least for a short time, a diminution of the intravascular pressure. Hypodermatic injections of camphor and alcohol, although slower in their action, would also assist in antagonizing the deleterious effect of over-distension of the right ventricle by the air-embolus. In all of my experiments where electricity was used it had no effect whatever upon the heart, even when applied directly to the organ, hence it would be of no use to resort to this agent for the purpose of sustaining or re-establishing the movements of this organ in serious cases of air-embolism.

3. In all post-mortem examinations after sudden death from air-embolism two constant pathological conditions are always found present: 1. A comparatively empty state in the left side of the heart and throughout the entire arterial system. 2. Distension of the right ventricle and intense engorgement of the whole venous system. Both of these conditions are, of course, due to the presence of air in the ultimate branches of the pulmonary artery, which prevents the free transit of the venous blood through the pulmonary capillaries into the left side of the heart. As a direct result of these conditions, we have a diminution of the intravascular pressure in the left side of the heart and arteries, and a corresponding increase in the right side of the heart and the veins. The immediate and greatest danger arises from an accumulation of air and blood in the right side of the heart to such an extent as will arrest the ventricular contractions by over-distension before the equilibrium between the arterial and venous circulation can be restored by the removal of the air-emboli from the pulmonary artery into the general circulation. Even the most extreme antagonists to the lancet

must acknowledge the benefit which follows its use in similar conditions of the circulation caused by other pathological conditions. In severe cases of pneumonia where the circulation has been so much obstructed that death is threatened by venous engorgement and over-distension of the right ventricle, the free use of the lancet is the safest and most efficient means in equalizing the circulation, as by the abstraction of blood from one of the veins, the intravascular pressure in the veins and right side of the heart is diminished in the promptest and most direct manner. As the adventitious air in the pulmonary capillaries can and will be forced into the general circulation by the contractions of the right ventricle, and as the urgent symptoms will subside after this has taken place, it is of paramount importance to gain time by protecting the right ventricle against an undue amount of additional labor. Among the indirect remedies to accomplish this object venesection deserves the first place. I could refer to a number of the experiments where copious bleeding from the distal portion of the open vein was promptly followed by improvement in all the symptoms. Vulpian asserted that, after insufflation of air into veins, the contractions of the heart were restored after the organ had ceased pulsating, by the abstraction of blood from the sinuses of the brain and the veins of the neck. I have never succeeded in restoring the action of the heart, after its pulsations had ceased completely, by any kind of treatment; but, when the contractions have become imperfect from this cause, I know that removal of the cause of over-distension will restore the force and efficiency of the contractions. H. Fischer[1] sarcastically alludes to this valuable agent in the treatment of air-embolism as are medy from which only homœopaths would expect to derive any benefit. An expression of this kind concerning such a valuable remedy, and in this particular connection, must be looked upon as unscientific and contrary to well-established clinical and pathological facts. I must, therefore, insist that venesection from the distal end of the wounded vein will prove beneficial in all cases of air-embolism where venous

[1] Ueber die Gefahren des Lufteintritts in die Venen, p. 17.

engorgement and over-distension of the right ventricle constitute elements of imminent danger. If practicable, the bleeding should always be done from the distal end of the wounded vein while the proximal end is compressed or ligated. The advantages of doing so are obvious for the following reasons: *a*. The abstraction of blood is accomplished in the shortest possible space of time. *b*. The blood escapes in a large stream from a capacious vessel. *c*. No additional instruments are required, and the infliction of another wound is obviated.

4. Clinical experience and experimental research teach us that when a certain amount of air enters the right side of the heart, death invariably takes place in a very short time and cannot be prevented by any of the indirect methods of treatment. It seems to me that in such cases it would be legitimate and proper for the surgeon to resort to some procedure by which the air could be removed directly from the heart. To accomplish this object Fischer recommends that forcible expiratory movements should be excited by inducing coughing, sneezing, or vomiting, with a view that during the forcible compression of the thorax the aspirated air would be forced out through the wound in the vein. Against this advice the following objections may be entered: 1. The difficulty or impossibility of exciting coughing, sneezing, vomiting, or any other act on part of the patient when in a condition of collapse. 2. If during the forcible compression of the chest, the air is forced backwards, it will be just as likely to pass into other veins than the one through which it entered. 3. The difficulty of preventing the admission of more air during the forcible inspiration following the forcible expiration. 4. During prolonged forcible expiration the intravascular pressure in the veins and right side of the heart is greatly increased, thus constituting an additional source of immediate danger. 5. Forcible expiratory movements of the thorax, by compressing the heart, will be more likely to force the air onward with the venous current into the pulmonary artery where it will do the most harm by causing asphyxia from a sudden and extensive air-embolism in that vessel.

The only direct means of removing air from the heart consist

in puncture of the right ventricle and catheterization of the right auricle combined with aspiration. The experiments made by myself in this direction have demonstrated that puncture of the right ventricle with an aseptic-needle two millimetres in diameter is in itself a harmless procedure. When we remember that in the human subject the heart has often been the seat of more extensive injury without any immediate or remote ill effects, we must abandon the idea that slight injuries of this organ are necessarily fatal. Small aseptic wounds of the heart heal rapidly, and in the same manner as in any other organ of the body. The hearts which I show you were removed from dogs a few days to three weeks after puncture; and not in a single specimen are we able to detect any evidences of organic changes, either in the substance of the organ or its serous membranes. In most of the specimens the point of puncture is marked by a minute cicatrix, visible upon the surface of the visceral pericardium. In penetrating wounds of the heart hemorrhage into the pericardium and compression of the heart from this cause are to be feared, and constitute the only source of immediate danger. There is no plausible reason why in the human subject an oblique puncture of the right ventricle should be followed by more hemorrhage than in animals, and consequently I have no hesitation in recommending puncture and aspiration of the heart as a justifiable procedure in cases of air-embolism which would otherwise necessarily prove fatal. The question naturally arises: What symptoms indicate a resort to puncture and aspiration of the right ventricle? Two different conditions, as far as time and symptoms are concerned, may call for this operation. The puncture should be made as soon as possible after the entrance of air, in the event that the primary effect of the heart embolus has produced sudden over-distension and paralysis of the right ventricle, an occurrence which would be indicated by immediate collapse and partial or complete suspension of the heart's action. In such a case the direct withdrawal of air from the right ventricle, as soon as possible after its entrance, affords the only possible hope of restoring the pulsations of the heart by removing the cause of

the mechanical over-distension. In the second class of cases the patient has collapsed, but the heart has withstood the primary effect of the aspirated air. The heart's action is rapid and tumultuous, perhaps at times intermittent. The ear applied over the cardiac region detects distinct churning sounds. Respirations are rapid, and all symptoms point towards imperfect circulation and aeration of the blood as expressed by the pallid face and blue lips. Some of the air has passed from the right side of the heart into the pulmonary artery, and from the obstruction of the circulation through this vessel the right ventricle becomes more and more distended, the contractions therefore are less perfect and more frequent. At this stage of things puncture and aspiration of the right ventricle will overcome the most urgent symptoms by the removal of air and spumous blood, and enough time may be gained for the right ventricle to force the remaining air-emboli through the pulmonary capillaries into the general circulation, and the life of the patient is saved. These are the cases where puncture and aspiration of the right ventricle can be done with a fair prospect of not only relieving urgent symptoms, but of ultimate recovery. They are also more favorable from the fact that more time is afforded the surgeon in procuring and using the aspirator. The puncture should always be made in an oblique direction, from below upwards, so as to make a valvular tract in the heart for the purpose of preventing hemorrhage into the pericardium, and also for giving the point of the needle a direction in which it is least likely to injure the opposite endocardial lining. The left intercostal space, between the fourth and fifth ribs, about an inch and one-half from the margin of the sternum, is selected as the best point for making the puncture. The needle, thoroughly disinfected, is connected with the aspirator, and, as soon as its point is buried in the tissues, a vacuum is created in the aspirator, and the needle is advanced slowly until spumous blood is felt and seen to escape, when it is firmly held in this position, and the contents of the ventricle are withdrawn as quickly as possible. My experiments have satisfied me that I generally removed the needle too soon, not having withdrawn a

sufficient quantity of blood and air. In some of the experiments I followed the aspiration by blood-letting with marked benefit. Removal of the same quantity of blood directly from the right ventricle would have been productive of more good, as less air would have been left in the heart. The experiments on dogs have shown that these animals will always recover if the quantity of air injected into the veins does not exceed one c. cm. of air to each pound of its weight. Double this quantity must be considered a fatal dose. If, therefore, we can remove by aspiration only a portion of air directly from the right ventricle we may be able to maintain the action of the heart and respiration until the embolism of the pulmonary artery has been relieved by the passage of air from this vessel into the general circulation. When the presence of a foreign body in any other part of the body threatens to destroy life no surgeon would hesitate to make an attempt to remove it, even if the effort should be attended by an increase of the immediate risk. Air in the right side of the heart acts the part of a foreign body, and, when it destroys life, it does so by causing mechanical obstruction to the circulation. The timely removal of the air is the only rational treatment in all cases where simpler measures have proved inadequate in preventing a fatal termination. An aspirator in good condition should be on hand in every well-regulated hospital, and of ready access in cases of emergency. If air-embolism occurs during an operation the instrument should be used before the heart has ceased pulsating, when prompt action on part of the operator may obviate death in an otherwise hopeless case. The experiments on catheterization of the heart were undertaken with a view to simplify the operative removal of air from the right side of the heart. An aseptic catheter was introduced into the wounded vein and passed into the right auricle, and by aspiration air and spumous blood were removed. The result showed that some of the animals were saved from impending death by this simple procedure. The great danger attending this operation consisted in the tendency of the blood to coagulate within or around the distal end of the catheter, and death from the formation of a thrombus in the

large veins and right side of the heart. If the formation of a thrombus could in some way be prevented with certainty, catheterization and aspiration of the heart would recommend itself as the simplest and safest measure in the operative treatment of air-embolism.

Catheterization of the heart is not a new suggestion, as the introduction of a tube into the heart for the same purpose was recommended more than fifty years ago by Magendie. I should recommend the adoption of this procedure only in case when the air has entered through a wound in the internal jugular, and when the symptoms leave no doubt that death would be inevitable without direct removal of the air by aspiration. An aseptic Nélaton's catheter with an open end should be introduced into the wound, through which the air entered, and pushed as quickly as possible as far as the auricle, when the air and spumous blood can be withdrawn by the mouth in the absence of any other means of aspiration. Admission of more air is prevented by compression of the distal end of the instrument before and between the aspirations. The whole operation must be done as quickly as possible for the purpose of preventing coagulation of the blood. For the same reason an instrument of large calibre should be used.

In presenting to you the practical outcome of my experimental work in the form of these few suggestions on the operative treatment of air-embolism, I am fully aware that in your own minds you have decided that my labor has been in vain, and that the means suggested do not admit of application in practice. In reply, I will say that desperate cases call for desperate measures. When death stares us in the face we have not only a right, but it becomes our most imperative duty to resort to any plan of treatment which holds out the slightest hope of saving the life of the patient. For myself I am fully convinced of the safety and usefulness of puncture and aspiration of the right ventricle in grave cases of air-embolism, where simpler means have proved of no avail. When no aspirator is within reach I also believe in the propriety of catheterization and aspiration of the heart as a last resort in all cases of air-

embolism where death would surely take place without it. If by the adoption of either of these methods of direct treatment of air-embolism a single human life should be saved, I shall feel amply rewarded for the labor incurred in the preparation of this paper.

In conclusion, I beg leave to submit the following *résumé* for your further deliberation and discussion:—

1. The presence of adventitious air in the vascular system during life gives rise to air-embolism.

2. Each air-embolus constitutes a mechanical source of partial or complete obstruction to the flow of blood in the vessel in which it is located.

3. Aspiration during the inspiratory movements of the chest is the direct or exciting cause of ingress of air into a wounded vein or sinus.

4. Elevation of the head is the sole predisposing cause of the entrance of air in wounds of the superior longitudinal sinus.

5. In veins the predisposing causes consist in—

   a. Elevation of the part wounded.

   b. Pathological or anatomical conditions which prevent collapse of the vein when it is wounded.

6. Insufflation of a fatal quantity of air into a vein produces death by—

   a. Mechanical over-distension of the right ventricle of the heart, and paralysis in the diastole.

   b. Asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery.

7. Insufflation of the same quantity of air into arteries is less dangerous than when introduced into veins. When death is produced in this manner it results from—

   a. Acute cerebral ischæmia.

   b. Secondary venous air-embolism.

   c. Intense collateral engorgement of the vessels of the brain and spinal cord. The manner of death being

determined by the amount of air injected, and the direction in which the injection is thrown, as well as the time which has elapsed between the operation and the fatal termination.

8. Air injected into arteries is readily forced through the systemic capillaries into the venous circulation and right side of the heart by the powerful contractions of the left ventricle.

9. Air-embolism of the pulmonary artery is relieved in a comparatively short time, provided the contractions of the right ventricle continue unimpaired for a sufficient length of time to force the air through the pulmonary capillaries into the general circulation.

10. The prophylactic treatment consists in proximal or double compression, or ligation of the vein which is endangered by the operation.

11. The indirect treatment has for its objects—

a. Prevention of admission of air.

b. Administration by inhalation of hypodermatic injection of cardiac stimulants.

c. Venesection.

12. The direct or operative treatment by—

a. Puncture and aspiration of the right ventricle.

b. Catheterization and aspiration of right auricle, which are proposed with a view to obviate the direct cause of death by the removal of air and spumous blood, thus relieving directly the over-distension of the right ventricle, and, at the same time, to guard against a fatal embolism of the pulmonary artery.

13. The results obtained by experiments on animals warrant the adoption of the operative treatment of air-embolism in practice, as a last resort, in all cases where the indirect treatment has proved inadequate to meet the urgent indications.

DISCUSSION.

Dr. J. Collins Warren, of Boston, Massachusetts.

I do not know that it is necessary to say anything in regard to the admiration which we all feel for the arduous labor of Dr. Senn. The work is of a character that we should value and cherish. We have too little of this kind of work in this country, and should be glad to see more of it.

In regard to the lesion of which he treats, I think that the criticism may be made that it is rather obstetrical than surgical. There are few members of this Association who have met with such an accident that has called for any exercise of their surgical skill. A certain amount of aspiration of air into veins has been seen by most every surgeon, but alarming symptoms are of rare occurrence. The cases are not numerous in surgical literature.

According to the investigation of those who have studied this subject beside Dr. Senn, it appears that the lesion amounts to a blowing up of the right side of the heart, so that its action is paralyzed and it cannot contract. Various other theories as to the cause of death have been brought forward, such as anæmia of the brain and air-embolism of the lung, but it seems to me that the lesion described is sufficient to explain the result. The heart undertakes to force out of it that which it cannot squeeze or compress, and it is paralyzed in its efforts to do it. It has been compared by some to fat-embolism. We know that fat-embolism, which sometimes follows surgical injuries, may be present and not produce any symptoms. The capillaries of the lung may be filled with fat-emboli and yet no symptoms be produced. At autopsies a large amount of such deposits are sometimes found without their existence having been suspected during life. One lung may be completely obstructed by disease, and yet the patient be in a condition to live, as far as the lung itself is concerned. It does not seem probable that the presence of air in the lung is necessarily fatal. It is the heart and the large vessels which bring the blood to the heart that are blocked, and circulation in them is stopped.

Experiment has also shown that a large quantity of air can be injected into the circulation if it is done slowly. We have then

frothing of the blood, but no mechanical obstruction to the heart, without embolism in the lung and arterial circulation, and without any important or dangerous symptoms resulting therefrom. Dr. Senn could tell us how large quantities can be injected in the horse without producing bad effects.

I presume that this condition occurs in a large number of surgical cases. I remember performing tracheotomy on an adult female, in which the superficial veins were opened. I could hear the clicking of the air in the wound with each inspiration. I paid no attention to it, and no bad symptoms resulted. I presume that it can only be of serious import when some large sinus like the internal jugular vein has been largely opened, or if the longitudinal sinus or the axillary veins have been opened, and a large quantity of air has been sucked in by a sudden inspiration.

In speaking of the axilla, I may say that I have noticed aspiration of air into the axilla, which for the time being disturbed me, as it simulated somewhat this trouble. In the pumping efforts of the thorax, air may be drawn into the loose connective tissue of the axilla, producing a sound resembling that of the entrance of air into the vein.

Dr. Senn stated that this accident was more common in surgery before the days of anæsthesia than since. He ascribed that to the erect position favoring the entrance of air. It occurred to me that there might be another way of explaining the fact, if such it be, and that is, that the tension which was placed on the muscles of the patient under any painful operation and the gasping efforts at particularly painful periods, might impel the individual to take a sudden, short, sharp inspiration with all the muscles pulling in such a way as to open the veins and draw in several large bubbles of air. I should think some such explanation as that quite as possible as mere posture.

As I have said, it is in obstetrical practice that we meet with this accident in all its hideousness. In preparing for this discussion, I was reading over an interesting report on this subject by one of our medical examiners, Dr. F. W. Draper. Several cases of air-embolism following attempts to produce abortion have occurred in Boston. One was that of a woman several months advanced in pregnancy who went to an abortionist. She sat on a bed while he introduced an instrument into the uterus, and then passed a syringe

and began to pump in air, it is supposed. Air was forced in at any rate. In about fifteen minutes, she gave a low moan, gasped once or twice, and in half a minute was dead. At the autopsy, the right side of the heart was distended with air. The ascending cava was also filled with air. Air was found not only in these positions, but also in the capillaries of the lung and of the arterial system, so that when the sternum was cut through, air bubbled out from the arterioles of the anterior mediastinum. It was argued in that case, that, as the air had gone so far, it might have been possible that it had been introduced with a vaginal douche previous to coming to the abortionist's office. That was disposed of by the statement that the circulation makes its round in a few seconds, and air can be rapidly distributed in a short space of time. As Dr. Senn has mentioned, we have in such cases, conditions most favorable to the introduction of air into the circulation. A small amount of air gets into a uterus which is relaxing and contracting, there is a certain amount of hemorrhage with clots which may cork up the mouth of the uterus and prevent the escape of the air. Under such circumstances there would be a large number of holes, communicating with veins which have no valves, and affording a ready egress of air from the uterine cavity. In addition, we have the action of the thorax which tends to aspirate the air into the circulation. Therefore I say that this accident is more likely to occur in obstetrical than in surgical practice.

In regard to treatment, it seems to me that such a paper as Dr. Senn's is valuable in showing us the mechanical mode of development of such condition, and in showing us the conditions most favorable for its occurrence, and consequently telling us more how to prevent the accident than to help the patient after it has occurred. As one writer has said, the only function of the physician in such a case is to be present at the autopsy and confirm the diagnosis. That appears to be his sole duty.

Theoretically, aspiration of the heart seems to be indicated, but practically there probably never would be such a consensus of circumstances which would enable us to be on hand, to have the necessary instruments, and be quick enough in our diagnosis to enable us to use the instrument instantly. Such an accident is like a stroke of lightning. The patient is dead the moment the condition is recognized. The heart has been aspirated in other condi-

tions, and I suppose it could be in this. I do not see that there is much ground for comfort in this reflection, but in this, as in many other conditions, we should hope by a more perfect knowledge to avoid them, rather than to cure them.

Dr. Edward M. Moore, of Rochester, New York.

Mr. President, I should like to introduce to the Association, Dr. Gordon, of Portland, Maine, who will detail to us an extremely interesting case bearing on this subject.

Dr. Gordon, of Portland, Maine.

The allusion of Dr. Warren to Dr. Draper's case reminds me of an accident which occurred to me during the past winter, and inasmuch as we profit as much by our faults as by our virtues, it may be interesting to describe it.

In the hospital to which I am attached, the Maine General Hospital, I found, when I went on duty, a lady with pelvic abscess. Others had operated by aspiration, but had not found pus. A few days later, the development having gone on rapidly, I found no difficulty in drawing off a pint of ordinary pus. A week or ten days later, I did the same thing. In the mean time our house-surgeon had been changed, and a new one came on duty. I would say also that the aspirator used in the hospital was a bottle aspirator. Those who are familiar with it know that the pump will either condense or exhaust the air in the bottle, according as the tube is applied to one or the other nozzle. The new house-surgeon, who was not familiar with the instrument, attached the tube at the wrong end. I was attending to the needle, and entered it at exactly the same point as in the previous operations. When I was ready, I said, "turn on." He did so, and at the same instant the woman gave a scream, put her hand over her heart, and in half a minute was dead. The fact was that the bottle had been charged with air, the aspirating needle was introduced, I simply made the connection between the needle and the bottle, and the result followed.

Exactly what happened it is impossible to say. This cavity had been exhausted largely by the previous tappings, and the air may have entered through some of the vessels in the wall, or the needle may have been passed into a vein. The air certainly got in. This is one of those cases in which there is nothing to be done. I have

only this to say, that I shall never use such an instrument where it is possible for any one beside myself to have anything to do with it.

Dr. J. Ewing Mears, of Philadelphia, Pennsylvania.

Was an autopsy made?

Dr. Gordon. There was not. The husband objected decidedly to a post-mortem examination. I think that there can be no doubt that death was due to air-embolism. The symptoms were those which belong to that condition.

Dr. Charles B. Nancrede, of Philadelphia, Pennsylvania.

I rise with mingled feelings of admiration and regret to discuss this paper. The admiration is for the indefatigable energy of our Fellow, Dr. Senn, and the regret is that I have nothing especially new to offer on this very important, and I think rather neglected, subject. It is true, as he stated in his historical account, that it had been long known to physiologists that injection of air into a vein in sufficient quantity was fatal. The original idea was that a single bubble was fatal. The first recorded case is usually stated to be that of Beauchesne in 1818. It is said that the elder Larrey had frequently observed it on the battle-field from ball or sabre wounds, I cannot say which, but more likely the former. My authority for the statement is a systematic work on surgery, in which it is stated that Larrey said to Brodie, when looking at the picture of a man with a wound in the neck: "Ah! I know how that man died. I have frequently seen them die from aspiration of air into the veins on the battle-field." If my authority is reliable, Larrey antedates Beauchesne in his observations, although Beauchesne was the first to *record* this accident.

I think that it should always be borne in mind in performing experiments that animals are not human beings. I am free from any prejudice against experiments, for my graduating thesis was an experimental one, and I had the honor at the meeting in Cincinnati of laying before the Association the results of my experiments on inflammation in animals. As Dr. Senn mentions, death occurs in some animals with greater rapidity and ease than in others. In the horse it requires a large amount of air to produce a fatal effect. He explained this as being due to the strength of the right ventricle. There is an observation of Bérard which explains it better, especi-

ally if you bear in mind some other observations to which I shall refer. This author states that the pulmonary capillaries in the horse are much larger than in other animals. There are other experiments which show that if there is one thing pre-eminently necessary for free capillary circulation, it is a proper density of the blood. It was not mentioned in the portion of the paper read by Dr. Senn, that the injection of water is capable of producing symptoms identical to those produced by the injection of air, although, presumably in the former case, they pass away. The only explanation of this is a sudden alteration in the density of the blood preventing it from circulating in the capillaries of the lung. I think that the observation of Bérard in regard to the size of the capillaries of the horse, in connection with the fact that we can inject relatively large quantities of air into the veins of these animals, and that the air is found in the arterial circulation, showing that spumous blood will pass through the capillaries of the horse, is most important in connection with the therapeutics of this condition.

I cannot think from the history of the cases and from the experiments on animals, that death is a simple matter in air-embolism. It is due to a complication of causes, the chief being, I think, anæmia of the centres of respiration. Dr. Senn and other experimental observers have thrown large quantities of air into the heart, much larger than could be introduced in an operation before fatal symptoms would come on. Undoubtedly, in experimental cases, the heart is distended with air, but this does not obtain in the accidental entrance of air into the venous circulation during operation. The heart has been found over and over again not distended with air. It has been found to move with great force and strength even after respiration has ceased, so that death does not come from the heart, but from respiratory failure due to deficiency of arterial blood in the centres of respiration. Undoubtedly the inability of the right ventricle to act properly is due not only to the mass of spumous blood, which it is unable to pass on through the lung capillaries, but also to the fact that the changes in the density of the blood alters its physical conditions, and the valves cannot be forced back as they would be by dense blood. The result is that the blood is thrown backward and forward from auricle to ventricle. Under such circumstances the removal of spumous blood from the heart may be valuable.

Dr. Senn has called attention to the chief facts, which are ex-

planatory of the conditions favoring the entrance of air into the venous circulation, but there are others in operation which he does not note, such as the position of the neck or arm, stretching the axilla in the various operations required in that neighborhood. A slight amount of tension will keep a vein open, so that even the contractions of the platysma myoid muscle will do so to a certain extent. Traction on a tumor will do the same thing. There is another rare condition which it is possible to conceive of, viz., the division of a vein in the angle of a wound. As you raise the flaps you hold that vein open and effect a temporary canalization, and the evil results that follow this.

Dr. Senn made the remark that in cases where air was supposed to have been generated in the blood, the observation had been inaccurate, and that all the cases recorded were those in which free hemorrhage had taken place, and in consequence there were open vessels where air might have entered. I dissent from that. There are a certain number of cases where that explanation does not hold. I had a case of my own which I cannot explain in any other way, and there are a number of others on record, occurring chiefly in the course of septicæmia. I had a patient whose arm I had amputated at the shoulder-joint. Eight days later he sat up in bed apparently pretty well. He gave a sudden exclamation, and before the nurse could reach him he was dead. I could get no post-mortem examination. There was nothing that would kill him so quickly, except air-embolism. Deep pressure on the stump forced out bubbles of offensive gas. There are similar cases on record.

As Dr. Warren has said, the preventive treatment is more important than the curative. We cannot carry our whole armamentarium around with us, and we are not likely to have an aspirator convenient. I have always had a great dread of air-embolism, and there are certain things that I invariably aim to do in operating on the axilla or neck. Thus, I think that complete anæsthesia is most important. The sudden inspiration after a painful cut undoubtedly tends to draw air in. In passing, I would allude to the fact mentioned by Dr. Warren in reference to the sucking sound often heard during tracheotomy. I have heard this sound frequently, and have been in times past much alarmed at it, but I have come to the conclusion that it is due to the opening of the deep layer of the tracheal fascia. I have heard this sound when there were no open veins.

It is probably due to the same conditions which produce a similar sound when air enters the axillary tissues. Where anæsthesia is not used the arms and chest should be firmly bound down so that no sudden inspiratory movement can be taken. In removing a tumor from the neck, I usually tie its deep attachments, or hold them with clamp forceps on the cardiac side, before twisting off the tumor. If it is possible I never make a cut where there is any tension of the cervical or axillary tissues, unless there is a finger on the cardiac side of the vein.

Should air enter, what can be done? Experiment has shown that artificial respiration in those cases where a non-fatal quantity of air has been injected is of service. This should be done in the recumbent position. Air will not enter as readily in the recumbent position as in the erect position. The head should be dependent, the limbs raised, and artificial respiration carried on. Treves has recently suggested a method for preventing further entrance of air. It may not be easy to tie the vein, but a wet sponge can be at once squeezed, filling the wound with water. Then, as has been shown by experiments in animals and in the human subject where recovery has taken place, the symptoms are partly those of syncope. The arterial blood should be diverted from the extremities to the brain by compressing the axillary and femoral arteries. This expedient probably also effects another purpose. It increases the intra-cardiac pressure so that it is possible to have the valves closed and the blood forced through the pulmonary capillaries.

It is hardly necessary to say that the cardiac action should be kept up by stimulation. There is one suggestion that I should like to make, viz., in addition to the subcutaneous injection of ether and the application of ammonia to the nostrils and so on, we should use subcutaneous injections of atropia. If it is true that the injection of larger quantities of air are required to kill a horse, because of the larger size of the capillaries in that animal, the importance of dilating the human pulmonary capillaries to their fullest capacities is seen. This is a secondary result of a large dose of atropia. It also stimulates the respiratory centres. I have seen it do it. I reported a case last year in which the pulse and respiration had ceased for about three minutes. It was a case of brain abscess. I attributed the temporary recovery to the injection of atropia, life being prolonged for six days. It is indicated, and it can do no

harm. It is true that atropia does depress the action of the heart a little, but you get more than a compensating advantage by the enlargement of the pulmonary capillaries. Experience has shown, that if the patient can be kept alive for a short time he may recover. After recovery the patients are apt to be attacked with secondary pneumonia, which is to be treated on general principles.

Dr. Senn. I am under great obligations to the gentlemen who have so ably discussed my paper. Their remarks bear evidence of study and careful preparation. In view of the numerous well-authenticated cases of sudden death during surgical operations it requires no argument to prove that the subject of air-embolism should be as important to the surgeon as the obstetrician. The very fact that so many surgeons have witnessed this accident is a strong argument in favor of discussing this subject at this time and in this place with a view of arriving at some definite conclusions pointing towards preventing this occurrence, or in case a dangerous, or fatal amount of air has entered, what measures might be resorted to to modify or annul its deleterious effects. The introduction of a few bubbles of air does not produce death, because it neither interferes with the passage of blood through the pulmonary circulation nor with the perfect mechanism of the heart's action. When air is introduced slowly and in small quantities, not sufficient to arrest the heart's action, it is forced through the pulmonary capillaries into the left side of the heart and arterial circulation, where it is brought into contact with an extensive surface for absorption, and by its gradual removal sudden accumulation in dangerous localities is prevented. A clicking sound heard during an operation is no indication that air has entered a vein; such a sound may attend the ingress of air into any hollow space. The sound produced by the entrance of air into a vein resembles a sucking sound, which is best expressed by the German word "schluerfend." Muscular contractions always produce compression of a vein, and thus would rather tend to prevent, than to produce, entrance of air. When the necessary conditions are present to maintain the patency of a vein after incision I consider the elevated position the most important element in causing the entrance of air. The experiments on the superior longitudinal sinus reported in my paper sufficiently corroborate this statement. In regard to the treatment by direct aspiration of the right ventricle, which saved a good percentage of

the animals where a fatal dose of air had been administered, I must again emphasize the fact that over-distension of the right ventricle occurs as much from accumulation of venous blood from the obstruction in the pulmonary capillaries as from the presence of air, hence any procedure which can remove the cause of the over-distension in the shortest space of time must be relied upon in preventing paralysis by over-distension. The all-important indication presents itself to maintain the action of the heart until the obstruction in the pulmonary circulation has been removed. Death does not take place instantly. The heart often continues to contract feebly for some time, until by accumulation of blood in the right ventricle over-distension results in arrest of muscular contractions. As aspiration of the right ventricle in itself is not attended by any immediate or remote danger, I must insist that it should always be resorted to in cases of air-embolism when other measures are inadequate to prevent death by over-distension and paralysis of the heart. Removal of air from the right side of the heart by aspiration also constitutes the most direct measure in preventing death by asphyxia or acute cerebral anæmia, as it diminishes the number of air-emboli which otherwise would subsequently pass into and obstruct the pulmonary vessels. The experiments have also shown the usefulness of venesection in these cases, the beneficial effect being due to its direct effect in preventing engorgement and distension of the right side of the heart.

Air-embolism produces obstruction in the pulmonary vessels by bubbles of air becoming arrested in the vessels, which act, for the time being, like any other kind of emboli by mechanically interfering with the circulation. In the horse the contractions of the right side of the heart, as compared with other animals, are more powerful, and on this account over-distension is not as likely to take place, the air being forced rapidly through the pulmonary capillaries into the left side of the heart. Injections of air into the left side of the heart are less dangerous, because the powerful contractions of the left ventricle are more competent to force the air from the heart into the peripheral circulation and through the systemic capillaries. A simple alteration in the density of the blood is not attended by such grave symptoms indicative of obstruction in the pulmonary circulation as we observe it after air-embolism. Intravenous injection of a saline solution after sudden

and severe hemorrhage is never attended or followed by such a train of grave symptoms as we observe after the entrance of any considerable quantity of air into a vein. Auto-transfusion, as suggested by Dr. Nancrede, would, as he states, increase the intracardiac pressure, and on this account I should be afraid to increase the function of an already over-distended organ. The best possible method to remove the cerebral anæmia is to diminish the intracardiac pressure, as by doing so we maintain the action of the heart until the obstruction in the pulmonary vessels is removed, and the equilibrium between the arterial and venous circulation is restored. Theoretically the use of atropia would be indicated; practically, however, I should expect a more prompt effect from the inhalation of nitrite of amyl.

In closing my remarks I desire to thank the gentlemen for the interest they have manifested in my paper, and for the many valuable suggestions made.

THE ETIOLOGY OF TRAUMATIC TETANUS.

By P. S. CONNER, M.D.,

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Known from the earliest times, a wound-complication of extreme gravity, the subject of many investigations and innumerable papers, traumatic tetanus is an affection respecting the cause and manner of production of which there is still much uncertainty, and little, we might almost say no, definite knowledge. There is therefore full justification for a brief consideration of the various views that are held respecting its etiology; the more so, as not only is there much advantage in knowing such respecting any disease, but, further, in this affection the adoption or rejection of certain methods of treatment is largely based upon the views entertained as to the cause of the muscular spasms.

The study of any considerable number of reported cases, in which the diagnosis has been correctly made, will quickly convince us that under the generic head are embraced two species, the one characterized by spasms primarily affecting the injured part, the other by those at first located in the muscles whose nerves have origin in the medulla oblongata. At least since the time of the appearance of Colles's paper (1852) there has been, and we believe properly, a disposition on the part of most to regard as cases of true tetanus only those of the last-mentioned kind. And yet a death-producing generalization of spasms originally local does, though rarely, take place, and the later stages of cases of this sort are identical with those in which the earlier symptoms were connected with the muscles about the jaws.

There is little or no question but that the seat of the disease

is in the central organs of the nervous system—scarcely more that it is essentially an exaltation of the reflex irritability; but how is this condition brought about? Is it by the nerves or the blood-current; by extension of inflammation, by reflected irritation, by septic changes, or by the presence and development of micro-organisms? Each of these theories, with the exception of the last, has had numerous advocates, and the arguments therefor have had assumed substantial basis in the symptoms present, the results of treatment, and the pathological conditions discovered upon autopsy. That it is by ascending neuritis that the morbid state of the cord is induced is thus indicated. There is beyond doubt nerve injury in the original wound, such injury being one either of incision, complete or partial, of contusion, of laceration, of puncture, or of compression or irritation from an unremoved foreign body. Resulting therefrom is inflammation of the nerve-cord, either involving its whole extent or, macroscopically viewed, affecting parts here and there as it is traced upwards, at times attacking few fibres and discoverable only by careful microscopical examination; such inflammation being indicated by congestion, by exudation, by connective-tissue proliferation, by degeneration of the tubules. Passing to and up along the cord, there are to be found such conditions of the bloodvessels, the coverings, and the gray and white matter as are indicative of inflammation. The chain would seem to be complete, and the correctness of the theory established; but nerves are constantly being injured, yet tetanus is of rare occurrence. In very many cases it has been impossible to discover either neuritis or myelitis, and it seems much like begging the question to claim, as has been done, that in such cases there is pathological change, but of so delicate a nature as not to be discoverable by our present methods of investigation. In the quickly occurring and rapidly fatal cases there is not time for the development and extension of inflammation. Inflammatory nerve changes have often been observed when there has been no tetanus, and the symptoms of acute ascending neuritis are altogether different. The spinal cord changes in certain cases are much more probably effects than causes, due to circulatory

states consequent upon the convulsions; in other cases they cannot be distinguished from those repeatedly found associated with diseases unattended by spasm. And, lastly, were an ascending myelitis the determining cause of the irritability, there ought to be observed a progressive involvement of the muscles upwards from the level of the injured area, which is not the case in true tetanus, or from the feet and legs, if it be true as claimed (Michaud) that no matter where the wound is located the most marked pathological changes are in the lower part of the cord.

Fifty years of experience has but confirmed the truth of Travers's declaration, that "The evidence to establish that tetanus has its origin in inflammation of nervous structures has failed."

If not of inflammatory origin, is tetanus a reflex neurosis, consequent upon remote nerve irritation? Such is the opinion most generally entertained, whether the disease be associated with a wound or occurring idiopathically. It is argued that, as every one knows, peripheral irritation will give rise to movements affecting, it may be, a whole extremity, movements independent of and in spite of the action of the will; that nerve-injury and pressure is often connected with localized muscular cramps or jerkings; that the disease is most frequently developed in cases in which, from the character of the wound, the nerves of the part are torn, bruised, or crushed; that not seldom it is found that an irritating foreign body remains in the tissues, and the sharper, the rougher, the more irregular the substance, the greater the likelihood of the development of the affection; that the state of abnormal reflex excitability especially occurs when the wound is in a part in which sensitive nerve-fibres are most numerous and of the highest functional value; that it is often so quickly developed that it could be so only through reflex irritation; lastly, that it can be experimentally produced by wounding nerves. By reflex irritation may properly be explained the manner of the production of the muscular spasms in those cases in which they originally affect the injured part, and thence pass upwards and outwards to ultimately become

general. Such progression is strictly in accordance with Pflüger's laws, and were the cases presenting the phenomena of it other than exceptional ones, perhaps not to be justly considered as of true tetanus, the theory of radiation from an irritated nerve-centre might without hesitation be accepted as the correct one. But what is known respecting the physiology of the nervous system does not warrant us in believing that in a healthy individual a reflex irritation starting, it may be, in the foot or the hand, passing over all the intervening portion of the cord, will produce in its upper part an influence that will manifest itself, not in a single or a few eclamptic seizures, but in a persistent contraction of the muscles, passing from above downwards to, sooner or later, in most cases, become general. If nerve-injury is the determining cause, why is the disease so infrequent, attacking but one in 200, 300, 500, 1000 of the surgical patients in large general hospitals, or of the wounded of modern wars? surely the assumed exciting cause is often enough present. Consider for a moment some of the special nerve-injuries which have been followed by tetanus. In a few cases examination of the wound has shown nerve-fibres included in the loop of a ligature—similar inclusions of nerves of smaller or larger size must have occurred over and again. In how many of the declared cases of tetanus must be seen either spasms beginning in the neighborhood of the wound or a mere association of nerve inclusion with true tetanus otherwise produced? Even if the freeing of the constricted nerve has put a stop to the morbid condition, in some of the cases at least study of the reported symptoms will show that there was present only limb spasms, tetanoid, but no more the convulsions of tetanus than are the stump-twitchings which are so often met with. Again, hand, and especially foot wounds that have unquestionably been oftentimes followed by tetanus, have they the danger attaching to them that is so generally believed? and, if so, is it due to the fact that in these regions are terminal fibres of highly sensitive character correspondingly liable to become irritated? Although it would seem from published statistical tables (*e. g.*, that of our distinguished Fellow, Dr. Yandell), that hand and foot wounds

furnish about 50 per cent. of the cases of tetanus that occur, before concluding that such tables establish the excessive danger of the injuries of these territories, errors of diagnosis must be eliminated and determination made of the number of these wounds in comparison with the whole number of traumatisms occurring. It is in this connection an interesting fact that in over 16,000 wounds of the foot in our late war there were but 57 cases of tetanus, and in perhaps 12,000 or 13,000 of the hand but 37 cases.[1] Again, in punctures of both hand and foot, it has not been so much when they have affected the sensitive parts of the fingers and toes, but when there has been piercing of the palmar and plantar fasciæ that tetanus has been most commonly developed.

If the pressure or more active irritation of a lodged foreign body is the exciting cause, why should it so seldom exert its deleterious influence? and why should the experimental injuries inflicted upon animals so generally fail? In only a very few cases, as those of McDowell and Brown-Séquard, has it been found possible to produce the tetanic state. The results of operative treatment certainly do not lead to the acceptance of the irritation theory. Amputation has seldom relieved, and is favored by few or none at the present day. Nerve-section secured 10 favorable results out of the 16 cases collected by Letievant, but in at least one-half of the patients who recovered the muscles first affected were those of the injured part; and further, this operation has been performed in too small a number of cases for its statistics to have much argumentative weight. Nerve-stretching, according to Agnew's table, was followed by cure in one-fourth of the cases in which it was tried. But here again the two classes of spasms have not been separated, and further, in most, if not all the successful cases, "internal remedies were also administered." In one of the cases reported as cured by this operation, of which I have personal knowledge, twenty-

[1] [NOTE.—In the paper, as read before the Association, an error was made in the statement with regard to the occurrence of tetanus after wounds of the hand. The author stated that "not a single case" had occurred. The remarks made upon this point in the discussion are thus explained.—EDITOR.]

four hours after the stretching the spasms were still present in the opposite limb, and chloral and the bromides were steadily administered. It is an interesting fact to notice in this connection that of the 350 nerve-stretchings for causes other than tetanus given in Agnew's tables, not a single one was followed by tetanus. If such exception was not accidental, is in it to be seen additional evidence of the correctness of the irritation-theory, or was it due to the absence or very limited amount of inflammation in and about the wound, and the great care taken to secure, if possible, primary union?

The two facts that speak most strongly in favor of this theory of reflex neurosis are the excessively quick development of the morbid state in certain cases, and the influence of cold, acting either alone or in conjunction with traumatism. It is certainly hard enough to understand how reflex irritation from a lacerated wound of the thumb could give rise to a tetanus that should kill the subject of it in fifteen minutes from the time of the injury, but much harder to explain the occurrence on any other theory. Unfortunately, Robison's case is very imperfectly reported, so that there is no way to determine whether or not the facts were as stated, and, still more, whether or not very material facts have not been omitted. The influence of cold has long been believed to be a potent one in the production of tetanus; especially when the low temperature has been associated with much dampness; such influence being most likely to be manifested in regions or seasons in which there is a great difference between the midday and midnight heat. Acting locally, upon an exposed wound, or generally, upon the body at large, it has been held that the cold damp air effects the terminal nerve-filaments, by reflex action arousing an abnormal irritability of the central organs. This may be so; and a like action may result from the high temperature of tropical countries; but, as will be seen later, the produced effect may be in consequence of the presence of chemical or living irritants, the development of which is favored by the thermal conditions, aided, it may be, when cold is acting, by the internal congestions consequent upon the external chilling.

If neither through nerve-inflammation nor reflex action is developed that extreme irritability of the central organs that manifests itself in tetanic spasms, how then can it be induced? Only by changes in their blood-supply; changes either in quantity or quality. Experimentally, convulsions can be produced by over-filling and by emptying, by injection and by bleeding, but such convulsions are not tetanic. Both an hyperæmic and anæmic state of the cord have been repeatedly shown upon autopsy, but here, as always, when diametrically opposite conditions are found, neither can with good reason be regarded as primarily causative. It is not then in the quantity but in the quality of the blood that there is to be found what is sought, if the humoral theory be the correct one, in the presence of a septic element developed in the wound or of a special organism coming from without. Neither chemical examination, microscopical investigation, nor experimental injection has, up to the present time, established the truth of this theory,[1] which is rendered probable, if at all, only by reasoning and analogy. That certain vegetable alkaloids and animal poisons entering the circulation so affect the spinal cord and the medulla as to give rise to severe and rapidly recurring, oftentimes fatal, convulsions is a fact. In the absence, therefore, of absolute proof to the contrary, it is not impossible that tetanus may be thus produced. In the wounds that are most likely to present this complication, there is, both in the fluids poured out and in the tissues disorganized, abundant material, in the decomposition of which may chemically be developed septic matter, that, under favorable circumstances of atmospheric conditions or constitutional state, may produce the tetanic spasms. Even in a physiological fluid, urine, Gautier was able to find a ptomaine capable of tetanizing animals. Does not the danger of punctured wounds of the fascia of the foot or hand lie in the pent-up fluids rather

[1] L. Curtis, of Chicago, thought that he had discovered in the blood a peculiar micro-organism, but further investigation showed that it was equally present in the blood of the healthy mother and sister of the patient, and in the water of a pond near by. Arloing and Tripier injected blood from a horse affected with tetanus into the jugular vein of a healthy one, but failed to produce the disease.

than in any nerve-damage, and is it not in the pus, the pieces of clothing, the bits of wood, that have been found in some of the reported cases, and not in the lacerated nerve-fibres that one should look for the exciting cause of the fatal affection? The veins and lymphatics of the nerves themselves may readily be the channels through which the morbific matter may be carried in, and the anatomical fact that a direct relation exists between the sensibility of a nerve and the number and closeness of connection of its associated lymphatics, may go far in explaining why tetanus is so frequently associated with slight wounds of parts endowed with high sensibility, as the fingers and toes.

The influence of climatic conditions and atmospheric states may find explanation in their power over decomposition and organic growth, or in the produced constitutional depression rendering the system less resistant to the poison or the germ. Because of such depression we may perhaps understand why malaria or scurvy or anxiety or grief have at times been found conditions precedent to the outbreak of tetanus. When no wound is present it can only be assumed that it is in the natural secretions that the irritant is developed. The idiopathic cases of the disease are the ones the occurrence of which it is hardest to explain upon the humoral, indeed, upon any theory.

But, it is contended, were the exciting cause chemical or organic, the disease toxæmic, its attacks should be associated with fever; which is not the case. Yet at times distinct prodromal malaise has been noticed, and exceptionally a marked elevation of temperature. Is fever a necessary result of the presence of poison in the blood or an essential symptom of a mycosis? For answer, we have but to remember the symptoms of strychnia poisoning, of hydrophobia, and of cholera. Perhaps, as was suggested by Dr. Joseph Jones, in tetanus the frequent absence of high heat has its explanation in the correlated muscular motion that is present.

From time to time reports have been made of a greatly disproportionate occurrence of the disease in a single ship of a squadron, building of a hospital, even bed of a ward. How

otherwise than by the presence and action of a virus or a germ can such be explained? That many wounded after a battle or long transport should be so attacked might with more or less truth be attributed to a generally acting atmospheric condition, but not any distinct localization, as it were endemic prevalence of the affection.

Is it not, then, from very careful chemical and microscopical examination of the secretions of the wound, the tissues in and about it, and the blood of the part and the system at large, that we may hope to receive definite information respecting that which gives rise to this obscure affection? Will it prove to be a peculiar toxic material developing in the breaking-down, dying and dead matter, or a specific organism that will, so far as its cause is concerned, ally tetanus with anthrax, septicæmia, and erysipelas, an organism entering into the open wound or carried in with the vulnerating body? Whatever it may prove to be, if it is discovered at all, we will hear no more of hypothetical "personal predispositions" or "parts of least resistance," and treatment will be directed either towards preventing the development of the poison, the entrance of the micro-organism, or the sustaining and quieting of the patient until such time as the one shall be eliminated or the other have completed its life-history.

DISCUSSION.

Dr. Edward M. Moore, of Rochester, New York.

I do not intend to discuss this paper, but I want to ask a question. In listening to the paper, which I did with much pleasure, I was hoping to have heard something in reference to the remarkable production of tetanus some two or three years ago, in various cities, among boys injured by the toy-pistol. In my own city a couple of days after the Fourth of July, there were seven or eight cases. In Buffalo there were more. In Baltimore, the same was true, and so in different parts of the country. There is a peculiar chemical factor here which it seems to me would throw some light on this subject, if

some one would investigate it. I would ask Dr. Conner if he can give any explanation of these cases.

Dr. Conner. The few cases of this kind that I have seen were cases in which a considerable amount of contusion and laceration was effected, and in which usually the wad was retained and the fluids pent up, and it seemed to me that the cases rendered more probable the correctness of the humeral theory. The only case that did not have tetanus was one which I saw about fifteen minutes after the accident. The hand was kept in hot water day and night until healing was effected. In that case, there was no irritation at all. I had one or two cases in which tetanus developed, but this was present when I saw them. These was nothing brought out at that time which proved one way or the other, but they served to make more probable the theory advanced.

Dr. Charles B. Nancrede, of Philadelphia, Pennsylvania.

I wish to make one or two remarks on a fact, for it seems to be a fact, of the difference that a few miles will make. It is stated that in one part of Long Island tetanus is well known, while a few miles distant, with apparently similar surroundings, it is practically unknown under any circumstances; whether it is due to some external agency, to some form of poison, or to the depressing influence of malaria, is not known. I would suggest to Dr. Conner that he investigate this matter. The statement will be found in a late work by Dr. Van Buren.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

Dr. Conner has alluded to the experiment in which blood from a tetanic horse was injected into another horse, not suffering from traumatism, without producing any effect. Unless a large bulk of the blood of the tetanic horse were injected, we could expect no different result. The tetanic poison, admitting it to be a poison of any shape, produces its effects more like strychnia, and its effects to a large extent depend on the quantity. We know that there are many cases of tetanus which run a mild course, and this is especially apt to be the case in the non-traumatic form. I recall the case of a boy, who fell, producing a slight abrasion over the lip. He had a pretty severe attack of tetanus, but recovered. I have had other cases which were purely traumatic which got well. We all know

that the so-called idiopathic tetanus is more apt to recover, and this is possibly due to the fact that there is less of the poison, whatever it may be, admitted into the blood. It is evidently not a ferment that multiplies itself in the blood. It is more likely a poison which produces its effects by a constant introduction of increased quantities into the blood. The experiment cited, it seems to me, would not definitely settle the question, unless done under circumstances that would effect the transfusion of a large bulk of the blood of the tetanic horse into the sound horse. In that way only could the experiment be decisive.

If I understood the tenor of Dr. Conner's paper, the conclusion reached, or rather to be inferred, is that the disease is due to a micro-organism, and I am inclined to adopt that view. I have long believed that it was due to some specific materies morbi in some way, and that it is not due to nerve or reflex irritation. I have never been able to satisfy myself that these theories would account for the facts, and have long held that it was due to some specific element of some sort. At the present day, we should expect it to be due to a micro-organism.

Dr. J. Ewing Mears, of Philadelphia, Pennsylvania.

I think we must all regret the absence of a fuller attendance, and wish that more Fellows of the Association were present that they might discuss this paper. If there is any subject which claims our attention more than another, this is the one I think that there is no time when the surgeon feels himself so thoroughly powerless than when he is brought in contact with a case of tetanus, and when it becomes his duty to treat it in hospital or private practice. I have seen a number of these cases, especially in hospital practice. I have seen tetanus follow wounds in the hand and foot, and I unfortunately saw one case in which it followed amputation of the arm, and the post-mortem examination showed that in this case it was due to inclusion of the median nerve in a ligature.

What we want I think in connection with this subject is a very full discussion of it. We want the opinions of surgeons and of the Fellows of this Association. Dr. Conner has given us his views in regard to the cause, which we need to investigate in order to arrive at a proper method of treatment.

I was interested in the statement that in the army there were very few cases of tetanus following wounds of the foot, and that in wounds

of the hand, no cases were reported.[1] If these records are correct, I do not know how the fact can be explained. I think that in the exposure to which the soldier was subjected after the receipt of a wound, possibly lying on the battle-field all night, in all conditions of weather, in rain and snow, we have the conditions which are commonly supposed to be important in the production of the disease. It seems to me very remarkable that there should not be one case of tetanus following wounds of the hand recorded.

I think that if we to-day should give our collective experience we could probably count some twenty or twenty-five cases that we have seen in hospital practice.

Dr. J. R. Weist, of Richmond, Indiana.

I did not hear the paper read, but I understand from the remarks of Dr. Mears, that in the records of the war there is not a single case of tetanus following injury of the hands reported. I remember one case coming under my own observation of gunshot wound of the finger in a private soldier from Maine. The finger was amputated and the man died within thirty-six hours of tetanus.

I have within the past two years had three fatal cases of tetanus from injury of the hand, in my private practice. Two were crushing railroad injuries, and in the other there was a crushing injury of the hand, in a child, from a window sash falling on it.

Dr. L. McLane Tiffany, of Baltimore, Maryland.

I was not so fortunate to hear the paper, and therefore, if I suggest things already mentioned by the writer, I trust that he will excuse me.

The experience of surgeons has been asked for. My observation has been confined entirely to civil practice. The most remarkable thing that has come under the observation of civil surgeons of late years, has been the occurrence of tetanus after wounds of the hand produced by the toy-pistol. In my own city, it has been so prevalent about the Fourth of July, as to necessitate the passage of an ordinance making it criminal to sell the toy-pistol. At one time, I think three years ago, there were in the nine or ten days following the Fourth of July fourteen deaths in Baltimore, the injury

[1] [See note, page 319.—Editor.]

being in the hand in all cases. A number were injured elsewhere in the body, but none of these died.

In civil practice, the prevalence of tetanus in the hot months and also its prevalence among the dark race, has inclined me to ascribe it to something more than reflex nerve injury. Reflex nervous diseases are much more rarely met with in the negro than in the white race. Tetanus is far more common in the negro than in the white. Burns of the head in the negro will produce tetanus more frequently than any other injury that I have met with. The explanations hitherto given have not been sufficient to satisfy me.

Dr. Moore. I would ask Dr. Tiffany the question which I asked Dr. Conner. I had alluded to the prevalence of tetanus after injuries with the toy-pistol, and I asked whether he had any explanation of this fact. Whether any chemical examination had been made. It is a marvellous fact that almost all, not all, but almost all of the wounds produced by this toy-pistol become tetanic. We have had Fourth of July operations for a good many years, and I recollect taking care of a little boy fifteen years ago, who had discharged a wad into his leg from a toy-cannon, but it was not connected with this peculiar kind of powder used in the toy-pistol. The patient had tetanus, but he recovered. He was kept for about two weeks under the influence of chloroform. In the cases produced by the toy-pistol I have not heard of a recovery. In Buffalo and Rochester we had some twenty cases, all of which died. I have seen the statement somewhere that some one had taken up the question and made a chemical examination.

Dr. Tiffany. At my request one of my associates looked into the matter of the chemical effect of the detonating powder, but he did not discover anything to account for the conditions arising.

I have not heard of a case of tetanus resulting from the toy-pistol that recovered. I have seen a number of cases of wound of the hand that did not have tetanus. I saw one notable case which was under observation two or three weeks, in which there was a wad and other substances under the skin which were discharged from the toy-pistol. These came out and the patient recovered.

Dr. Mears. I desire to say that I reported a few years ago a case of tetanus after wound of the hand in a boy who was injured by

the toy-pistol. At my request an examination was made by a chemist in Philadelphia, and the result was the same as obtained by Dr. Tiffany. Looking further for an explanation of the great mortality following wounds of this kind, I came to the conclusion that it was due to the character of the cartridge and in the manner in which it is placed on the pistol. In the pistols examined by me I found that the cartridge was placed over the nipple, and not in the barrel, and that it was exploded by the contact of the hammer directly upon it. When the discharge takes place it is broken into fragments, and the pieces formed puncture the palm of the hand. In the case under my charge, I found a piece of the cartridge in the hand. Therefore, finding no explanation in the composition of fulminate, I expressed my belief that the results were due to this peculiar character of the cartridge.

Dr. David Prince, of Jacksonville, Illinois.

I have seen one case of wound of the hand from the toy-pistol which recovered. It was found, at the commencement of the symptoms giving rise to anxiety, that a portion of the cap was imbedded in the cutaneous substance of the hand. The medical treatment had been under homœopathic management, and probably had been chiefly negative.

As regards the character of the cap, it probably contains too much powder or too little. If it contained more, the material would be driven into the fleshy portion of the hand, and there would be drainage. If there were less, it would simply produce a contusion. There is ordinarily just enough to imbed the cap in the corium.

The common opinion is that in the case of a wound of the foot produced by treading on a rusty nail, the best treatment is to make a free incision in order that there may be drainage and a free discharge of any material that is present. This is on the theory ordinarily entertained that the cause of tetanus is nervous and not bacterial. By the free incision we are supposed to get rid of the irritation of the nerves from pent-up materials. I have no theory upon the subject.

Dr. Nancrede. I can only recall the histories of eight or ten cases at the present moment which I have seen in my own practice and in that of others. The following were the causes: a bite of the thumb; a punctured wound by a harrow-tooth about the middle

of the leg, which had healed when I saw it; a wound, produced I think by a blow on the forehead; a compound crush of the finger requiring amputation; an operation by ligation of hemorrhoids; a compound crush of the hand from a blow from a block, in which partial amputation was required, and this was followed by gangrene; a compound comminuted fracture of the skull; and in another case there was some trifling injury.

I have seen only two cases of acute traumatic tetanus that recovered. In one the broken fragments of a fractured skull were removed, and large doses of hydrate of chloral given. The boy recovered, but for a long time had the risus sardonicus. The other case was one of amputation of the finger. It was reamputated, and the patient got well. I do not say that the amputation cured him. One of my colleagues amputated a limb for tetanus, but it failed to arrest the disease. I have seen the musculo-spiral—and I think the median and ulnar nerves—stretched, and the patient get rather worse than better. I have divided the nerves with the same result. I have kept the patient for nearly twenty-four hours under an anæsthetic, and when ether seemed to lose its effect I have changed to chloroform. I have seen Calabar bean administered until I think even toxic effects resulted. In fact I have seen almost everything which has been suggested tried without avail. Calabar bean gives relief. Chloral gives relief. Anæsthesia gives relief, but only so long as the patient is kept well under its influence. In a case of subacute traumatic tetanus I used inhalations of nitrite of amyl, and as long as they were used at intervals of about four hours the man was relieved, the spasms were mitigated, and he could take his food readily. On two occasions, owing to the insubordination of the assistant, who in my absence insisted upon changing the treatment, the remedy was stopped. On the first occasion the man got markedly worse, and on the second he died. The first effect was to produce increased spasms, but relief was soon experienced, and the man insisted that it did him good.

All the cases that I have seen have been after comparatively trifling injuries. In one case it apparently occurred after the wound was healed, which has been said to be impossible. I have about come to the conclusion that in acute tetanus the French maxim holds good, viz., so many cases, so many deaths. Neither of the successful cases just mentioned were under my own care.

Dr. Prewitt. I desire to say, in regard to the effects of the toy-pistol, that two years ago, I saw several cases of injury of the hand from the toy-pistol. None of these cases resulted in tetanus.

Two or three months ago I had a case which is certainly unique as far as my experience goes. A man received an injury of the arm from a threshing machine which resulted in tetanus two or three weeks later. I saw him a few days after the development of tetanus. I placed him on bromide of potassium and chloral with five-drop doses of Fowler's solution every three hours. This was kept up two weeks, and finally the man recovered. After continuing this treatment for a time he got into a sort of typhoid condition. The tongue was dry, he was delirious, and this continued for two or three weeks after the discontinuance of the Fowler's solution. There was no diarrhœa and no other symptom indicating the injurious effect of the Fowler's solution, unless it was the dry tongue. It was followed by paralysis of the flexor muscles of the left foot. He dragged that foot. This had improved somewhat when he had left the hospital. The injury had been on the right arm.

Speaking of the effects of Fowler's solution, I remember another case resulting from a punctured wound of the foot with a nail. A week or ten days later the man came stating that he thought he had lockjaw. I found that there was some stiffness of the jaw. I directed him to go home and go to bed. That patient also recovered under the use of bromide of potassium and chloral with five drops of Fowler's solution every three hours. He took this for ten days without any ill effect. I think that there are few persons who could take Fowler's solution so long without producing toxic effects in some way. It seemed to me that there was a tolerance to the remedy in these cases of tetanus which we should not have expected.

Dr. B. A. Watson, of Jersey City, New Jersey.

I regret that I am unable to add anything bearing specially on the etiology of this affection. It is a subject which I have recently examined, and although I have treated a number of cases, I am as much at a loss to-day with regard to the causes and treatment of the disease as when I saw the first case.

Dr. Moses Gunn, of Chicago, Illinois.

There is one idea which I presume has occurred to all. I have had occasion to see a good many cases of this affection, almost all

of which were fatal. I recall three cases of recovery. In two the recovery was perfect; in the other there was paralysis, the result of nerve stretching which seemed to be a circumstance in the cure. In the second case in which nerve stretching was done, the recovery was perfect. In the third case recovery followed amputation and the use of Calabar bean. This was eighteen years ago, when Calabar bean was first introduced.

The idea which I want to advance is this. It has been frequently remarked that those cases which recover are slow in their development, so that if the patient lives ten days recovery may be expected. Those cases which come on slowly are most apt to recover. I think that we may formulate this in this way—the fatality will be in direct ratio with the acuteness of the attack.

The PRESIDENT. I want to make one remark. In speaking of the etiology no reference has been made to predisposition. In my opinion there must be a predisposing cause. The system must be in a tetanoid condition. I have had several cases brought about by very slight causes. A young man came to see me about a small ulcer on the knee. He insisted on having the leg amputated, as he was going to be married and wanted to be in as perfect condition as possible. I declined to do it. He went to others, who also refused to amputate. Some proposed to excise the ulcer and draw the skin together, and he came back to me full of that idea. I thought there could be no harm in that, so I excised the ulcer and drew the parts together. This was on Friday. I was called into the country, and did not return until Tuesday. I had left the patient in the hands of another physician. As I was returning on Tuesday morning I picked up a paper, and the first thing I saw was a notice of the death of my patient from tetanus. In that case there must have been a predisposition, which was brought into existence by a slight exciting cause. I must suppose that there is a previous peculiar irritability of the nervous system, so that the slightest cause will develop the disease. If that does not exist the most serious injury will not bring it about.

Dr. CONNER. It is exceedingly gratifying to me to find that my paper has brought out so much discussion. The more that I have heard, the more I have thought of my own experience and studied the reports of others, the more convinced have I been that no nerve

theory will explain the conditions ; or, not to be so dogmatic, I will say that the humeral theory accounts for the greater number of cases.

The President has referred to predisposition. There may be such a thing, for we know that there are predispositions to various forms of affections. There is, however, one serious objection to this idea, which prevents me from attaching much weight to it. These individuals who have this predisposition, have had all sorts of injuries, and have not had tetanus developed; and only after a while is the individual in such a state, perhaps from a local cause, that it may be developed; yet the original, inherited irritability is present throughout the whole life of the individual who has been hurt a thousand times.

A good deal of attention has been directed to the toy-pistol. As far as my observation and reading go, there is nothing in the special character of the fulminate. The danger lies in the fact that it is a powerful one, and sufficient to drive the wad into the palm of the hand and even through the hand. It carries in a foreign body which quickly becomes saturated with fluids. The fluid is pent up, and it is easy for decomposition to take place in it and in the damaged tissues. It requires but little septic material to produce very disastrous results, just as it requires but a small quantity of strychnia to produce the tetanic state, followed by death.

Dr. Tiffany has referred to the fact that the negro is peculiarly subject to tetanus. An explanation of this may be found in the fact that the negro's secretions are peculiar. Of this there can be no question. It is an easy matter perhaps, to induce such decomposition in the secretions of the skin and the hair follicles as shall give rise to this trouble. Wounds of the negro's scalp are said to be frequently followed by tetanus, and here there is an abundant secretion. Dr. Tiffany has also called my attention privately to the fact that the negro is particularly liable to cerebro-spinal meningitis. Perhaps this too is due to the nature of the secretions of the scalp and the general secretions of his body and changes effected therein.

As far as operative treatment has gone, it seems to indicate that the humoral theory is correct rather than that the disease is due to an irritant. Sometimes a case gets well after amputation, but generally it does not. The cases that recover are those in which agents to allay irritability are given, the patient being kept quiet until the irritant can be removed, or sustaining treatment is em-

ployed until spontaneous elimination occurs. Active interference with nerves I have seen practised without success.

In regard to the wounds, I have seen more cases of tetanus occurring after major than minor injuries. In one case the left shoulder was crushed by a shot; in three cases it was after amputation of the knee-joint. In one case the wound was nearly healed. The individual was placed in a ward, the hygienic surroundings of which might have permitted the development of the poison. I have seen it follow a crush of the elbow, and took out a section of the nerve, without benefit. I have lost one case after operation for strangulated hernia, and know of one where it followed ligation of hemorrhoids.

According to the old and popular idea, the rusty nail does the damage. Rust does no harm. The nail does harm because it passes through a dense fascia, pressing the fibres aside, the fluids are retained, and decomposition is readily effected. Incision is of advantage, not so much because it relieves the tension, but because it allows the fluids to escape and the dead tissue be thrown off, and thus prevents absorption of the products of decomposition.

It seems to me that so far as slight injuries of the fingers and toes are concerned, the anatomical fact to which I have referred is an important one, viz., that the more delicate and acute the sensibility of the part, the greater is the number of lymphatics, and therefore if it is by the lymphatics that the material is absorbed, of course a wound of a nerve of high sensibility would be more likely to be followed by absorption than one of lower sensibility, the channels being more numerous.

I have drawn no conclusion, although I have an opinion upon the causation of tetanus. I hope that in the early future some of our workingmen will take up this subject, and further, if investigation show that it is due to a micro-organism, that this will be cultivated. Everything looks in the direction of a specific definite cause for each disease, and a number of diseases are almost certainly due to micro-organisms. I am sure that it is no more unreasonable to suppose that tetanus results from a microbe than it would have been ten years ago that erysipelas was dependent upon such a cause. I do not know that there is anything to indicate that it is an organism any more than that it is a ptomaine, but I believe that in one or the other will ultimately be found the answer to the question—What is the cause of tetanus?

An interesting point in regard to reported cases of tetanus is whether the spasms began in the wounded part, or whether they are cases of true tetanus with trismus as the earliest symptom. In the observations that are made, special attention ought to be given to the statement where the spasm began. I have looked over case after case, in the hope of getting some information upon this point, but have usually failed. I believe that it is right to make a broad line of distinction between the two classes.

REPORT OF A CASE OF CHOLECYSTOTOMY.

By C. T. PARKES, M.D.,
PROFESSOR OF ANATOMY IN RUSH MEDICAL COLLEGE, CHICAGO, ILLINOIS.

The rather recent revival of operative procedures for the relief of obstruction of the biliary ducts, has awakened a new and lively interest in all such cases; especially as the entire number of reported operations falls within a total of fifty.

Of this series, submitted to operation, by far the larger portion have been cases of cholecystotomy, by means of which a distended gall-bladder has been positively relieved, and in most of them the cause of obstruction, retained gall-stones, removed. In a few, no gall-stones were found, while in others the cause of the obstruction could not be remedied.

The excellent and exhaustive paper, based upon four cases, presented to the profession by Drs. Keen and Musser, of Philadelphia, portrays the operation of cholecystotomy, with all its vicissitudes and requirements, together with the symptomatology of duct obstruction, so completely and lucidly, that there seems but little more to be said; still, I am induced to bring to your notice the history of a patient, with gall-stones, who has been under my care during the past winter, and who was relieved by operation from their presence and the long-continued distress incidental thereto. Each one of these cases has some special circumstances of interest connected with it, and trustworthy conclusions only become possible after a consideration of all the facts culled from a large number of operations.

Mrs. K. B., aged 29 years, married, born in America, and residing in Chicago, came under my charge December 10, 1884. She had been made quite an invalid by years of suffering, and when

first seen by me had just passed through a two days' siege of great agony.

She related a history of more than two years' duration of suffering from paroxysmal pain, referred to the right hypochondriac and epigastric regions, and radiating beneath the right shoulder. These paroxysms were seemingly unattended with much or any febrile disturbance; were sometimes accompanied with constipation, and generally with loss of appetite. Quite often with them came nausea and vomiting. No biliary matter was ever vomited, and the feces were at all times light gray in color.

The attacks were of uncertain duration and somewhat uncertain frequency, but seldom remained absent longer than two weeks; so that the patient never had time to recover from the effects of one attack ere she was prostrated by the recurrence of another.

One year ago a gall-stone as large as a coffee bean came away by the bowel; no others were found previous to or since that time, though often sought after. No amelioration of any of her symptoms followed its extrusion.

The patient had been under the care of worthy physicians all this time; they all diagnosed the cause of the trouble to be the existence of gall-stones, and had subjected the patient to every known remedial medicine, without relief other than that coming from the use of large doses of opium, and even with these the patient's agony was intense during the acme of the paroxysm. Among the agents used were chloroform, ether, olive oil in large doses, and many other "cures," the best results following the administration of morphia.

Examination revealed the following facts: The patient is quite well nourished, flesh firm and in good quantity; the skin is slightly, yet noticeably jaundiced all over the body. There is no complaint of itching. Complexion rather pale and sallow; facial expression indicative of suffering and dread. Tongue clean and red. Examination of the thorax reveals nothing abnormal in the condition of its contents.

On inspection, the abdomen shows a clearly defined bulging of the right hypochondrium, extending into the epigastrium. Percussion defines liver dulness extending downwards fully three inches below the edges of the costal cartilages, also to the left some distance; again the dulness reaches to a higher point into the thorax than is normal. Palpation gives evidence of a resistant mass,

smooth, well-marked and slightly tender, evidently an enlarged liver. Careful circumscribed percussion seemed to establish the existence of a separate point of dulness, in area about the size of a silver dollar, near the centre of the edge of the liver dulness, and in a line with the tip of the tenth costal cartilage. This had resonance surrounding it on all sides, except above. It was somewhat elastic to the touch, and was supposed to be the fundus of the distended gall-bladder. No calculi could be certainly diagnosed as present by any external manipulation; subsequent results showed they would have eluded acupuncture; it was not practised. The feces are still light colored, both during and after the paroxysmal attacks of pain. There is no ascites or tympany, neither is there the slightest evidence of œdema of the lower extremities.

Chemical analysis of the urine fails to reveal any evidence of albumen or other abnormal condition, although chocolate colored; there was very slight reaction showing the presence of bile. The microscope gave no testimony in favor of degenerative changes in the kidney.

The prolonged existence of complaint, referred to the one place, with absence of any evidence of other trouble, either benign or malignant, confirmed the diagnosis already made of obstruction of the gall-ducts. Mrs. B. was told that no medical treatment would probably give her relief; that she had been faithfully and properly treated by her previous attendants, and that nothing could be added to what they had already done with medicine, but that there remained for trial the operation of opening the gall-bladder. After an explanation thereof, she immediately gave consent to have the operation performed. With that intent she entered the Presbyterian Hospital of Chicago, Dec. 15th, 1884. On Dec. 17th, a public consultation was held with Profs. Gunn, Ross, Etheridge, and Bridge, and Dr. Whitney, all of whom concurred in the diagnosis, and the probable relief from operation.

After preparation in accordance with the principles of strict antisepsis, in the clinical amphitheatre of Rush Medical College before the past winter's class and with the assistance of Prof. Moses Gunn and Dr. E. P. Davis, the following operation was performed Dec. 19th, 1884: An oblique incision, five inches long, was made over the tumor parallel to, and about two inches away from, the edge of the costal cartilage. The walls of the abdomen were slowly divided, until the peritoneum was exposed throughout the length of the wound; all

bleeding points were occluded with the snap forceps or ligature, before opening the peritoneum. When this was finally done, there was exposed to view only the smooth, even surface of the enlarged liver. The lower edge of this organ was fully two inches below the line of the wound. Nothing could be seen of the gall-bladder.

By passing two fingers into the wound and beyond the liver, the distended gall-bladder, round and tense, was readily felt. The finger was slowly and carefully carried around it, and by this means slight adhesions between it and the surrounding organs were broken down. Then the enlarged liver was forced up beneath the ribs by pressing upon its lower surface, and in this way the gall-bladder was brought into the abdominal opening. Considerable difficulty was experienced in retaining it at the opening, for every inspiration made by the patient forced the liver downwards and carried the gall-bladder with it. Prof. Gunn overcame this difficulty by placing his finger on the under surface of the organ and forcibly holding it up beneath the ribs. Two small sponges, with tapes attached, were placed to the right and left of the bladder, to isolate it as much as possible, and at the same time to absorb any waste that might fall into the abdominal cavity.

The walls of the gall-bladder were very thin, and the clear fluid which it contained could almost be distinguished through them. An aspirating needle of medium size was thrust into the centre of the fundus with the intention of withdrawing the contained fluid by aspiration. After its introduction some movement of myself or the patient accidentally displaced the needle, and immediately there followed a free flow of viscid mucus, slightly tinged with biliary matter. This was prevented from entering the abdominal cavity by drawing the bladder closely against the lower corner of the wound; a slight puncture with the knife was followed by an outflow of three or four ounces of fluid; none entered the peritoneal cavity. As soon as the viscus was empty its walls were seized by two snap forceps about an inch apart, and the opening in the bladder enlarged to that length. Gall-stones were then sought for, first by a probe, then with a pair of forceps, and finally with the finger.

The cavity of the bladder was thoroughly explored with all three, but no bodies of any kind were found in it. The edges of the wound in the gall-bladder bled quite freely, and were compressed here and there with forceps. The difficulty attending the great enlargement of the liver prevented as critical an examination of

the common duct as one would like; still, attempts were made to follow its course, but without success. No gall-stones were found, nor any other sources of obstruction. The wound was closed in the following manner: stitches were first introduced through the edges of the abdominal wound to the right and left of the point selected for the attachment of the gall-bladder in the establishment of a fistula. Then the opening in the gall-bladder was fastened to the edges of the central portion of the external wound by means of five sutures, first passed through all the tissues of the abdominal walls, and then through all the coats of the viscus; so that broad peritoneal surfaces were brought in contact throughout the entire circumference of the bladder wound. Great care was given to the security of these sutures, from the fear that the dragging of the heavy liver might displace them. A very large sized perforated rubber drainage-tube was carried to the bottom of the gall-bladder, and the sutures all fastened after thoroughly cleaning all portions of the peritoneal cavity in the vicinity of the wound. Iodoform was dusted on to the wound freely, and it was covered with an extensive and thick dressing of dry sublimated gauze. Outside of this was placed a mass of borated cotton, and over all the usual bandage.

The patient showed signs of considerable shock; was given a hypodermic injection of whiskey. There had been no attempt at vomiting. She was put to bed, head low, and hot-water bottles to feet. Reaction came on fairly well; on becoming conscious she complained of severe pain at the site of the operation; was fed cracked ice and given morphia hypodermatically.

At 10 P. M. the pulse numbered 84; temperature was $98\frac{1}{5}°$, and the skin was moist and cool. Complains of pain in wound at intervals; has some nausea, but has not vomited. Passed a comfortable night, and was free from nausea. The patient was now bolstered up into a half-sitting posture in order to relieve, as far as possible, tension on the gall-bladder.

After complete recovery from the effects of the anæsthetic the patient was quite comfortable, and until the wound healed the highest point reached in temperature was $99\frac{1}{2}°$. For a few days alimentation was mainly carried out by rectal injections; keeping the stomach quiet aided in preventing vomiting; had this occurred the attachment of the cyst would have been greatly endangered. Aside from nervousness the patient was free from any inconveni-

ence. The urine was dark in color and normal in amount. The bowels were moved on the third day by enema.

The wound was not disturbed at all, merely additional dressings of dry cotton placed over that already on, for the purpose of absorbing a very profuse flow of greenish fluid which showed itself on the second day, and rapidly saturated everything about the patient. On the fourth day, December 24th, owing to the profuseness of this discharge, a change of dressing became imperative. The wound was found glued together throughout its entire length, except at the site of fistula. No tenderness or swelling, indicative of any inflammatory condition, was present. The enlarged liver had entirely disappeared, and percussion placed it within the normal boundaries. The line of the wound, instead of being flush with the surface of the chest, was drawn under and beneath the costal cartilages. The free flow of greenish fluid had evidently entirely relieved the engorged hepatic ducts. Some of the stitches in the abdominal wound were removed. The drainage-tube was taken out, and the cavity of the gall-bladder probed; deep in its cavity some foreign body was felt, and two of the smaller stones here shown were removed by the forceps; no others were felt. The drainage-tube was replaced, and the same kind of dressings as first used reapplied.

On the seventh day, December 26th, redressing was again required, on account of the free discharge of similar greenish fluid, so abundant that it saturated the bedding as well as dressing. All sutures removed; wound entirely healed. Probing of the cyst cavity again discovered foreign bodies, and with the forceps five more gall-stones were removed. The last, the largest here shown,

Fig. 1.

was seized with much difficulty, and seemed to be embraced by mucous membrane (Fig. 1). No more stones were found at any time. The total weight of the seven stones removed is 7.22 grs.

The patient improved rapidly in general health; all the functions are performed normally; the jaundice has entirely disappeared; she is free from all pain and in bright spirits, up and about the

room. At the end of two weeks the patient could, apparently, have safely left the hospital; had she lived away from the city, she could have been sent home as a complete cure. The probe when introduced into the fistula seemed to come in contact with a roughened surface. The stools were still light-colored, unstained with bile. Every day or two a profuse flow of muco-biliary fluid came from the fistula, which the patient again said was accompanied with severe pain. The amount of pain was undoubtedly exaggerated, as the pain was not complained of until the discharge made its appearance outside of the dressing, and was on several occasions overcome by hypodermic injections of water.

Still all these occurrences seemed to point to continued occlusion of the common duct from some cause. Half a pint of sweet oil was administered with the hope of overcoming the obstruction. No stone was found in the subsequent dejections, but there came away six round, firm, white bodies, about the size of a bean, which were at first supposed to be concretions of cholesterine; examination showed them to be merely inspissated mucus and free oil. Perhaps such are the only bodies recovered after using sweet oil as a remedy for this trouble.

After failures to obtain relief with remedies, it was determined to again perform laparotomy to confirm a diagnosis of partial occlusion from stenosis of, or an impacted stone, close to the intestines, in the common duct. On January 18th, with the assistance of Drs. R. Tilley, E. P. Davis, and L. H. French, a vertical incision, four inches in length, commencing at the fistula, was carried through the abdominal wall. The deepest part of the dissection was made with great care, in anticipation of meeting with adhesions between the abdominal walls and intestines. These adhesions were not entirely avoided; near the gall bladder, at the highest point of the incisions, the walls of the duodenum were partially torn through and troublesome bleeding came from the tear, which was finally controlled by sponge pressure. The loose intestinal folds gave no trouble, and were easily held out of the way, so that the entire course of the duct, from the gall-bladder to the duodenum, could be readily and clearly traced with the finger. It felt like a dense hard cord. The bladder was entirely empty. No calculus could be found in any portion of the duct. Bougies of different sizes were passed through the fistula and guided by the finger; an attempt was made to pass them into the duct; no suc-

cess was met with. Then a steel sound, No. 11 French, was passed through the fistula, and again guided by the finger in the peritoneal cavity, after some manipulation, was finally carried into and through the common duct into the cavity of the intestine. No calculus was felt and no other special obstacle to its passage was recognized. The abdominal wound was thoroughly closed with sutures in the usual manner. No drainage employed; antiseptic gauze used for dressing.

The patient rallied well from the operation, but free vomiting soon showed itself, and the ejecta were mainly bile. This vomiting of bilious fluid continued during the first twenty-four hours after the operation, and there was no discharge from the old fistula. An enema brought a free discharge of bile from the rectum, the first to show itself since the trouble began, quite two years ago. All these facts proved that the passage of the sound had restored the patency of the biliary ducts. Some discharge soon came through the fistula, mostly mucus, but bile stained. The dejections were carefully examined while the patient remained in the hospital, but no gall-stones were discovered. Convalescence was without interruption, appetite returned, and pain was entirely relieved. The sutures were removed on the seventh day and the wound found completely united by first intention. The patient was discharged well, with the fistula rapidly healing on Feb. 1, 1885.

On April 1, 1885, two months after leaving the hospital, Mrs. B. sent for me, as she was again suffering from severe pain in her side, which had come on during the day. I found the fistula had been entirely healed for two weeks, and that she had been absolutely free from pain during the previous two months. The day before my visit she had been out all day exposed to very bad weather, and, as a result, had a very severe bronchitis, accompanied with pain and tenderness over the site of the old attacks. The fistula was reopened with a tenotomy knife and gave exit to quite a quantity of bile-stained mucus, which continued to flow during the next day. The pain was quickly and entirely relieved by this procedure. A small drainage tube was fastened in the fistula, and is to be retained with the intention of keeping it open for some time. Bile has not ceased to be discharged through the natural channels at any time since the last operation in February. The common duct is evidently too small in calibre to carry off any increased flow

of biliary secretion. The patient has improved greatly in general health, gained flesh, has a clear complexion, and has resumed all her household duties.

Some suggestions follow as an outgrowth of this case which are of practical importance and application.

It is quite certain that the gall-stones removed from this patient were not lodged in the gall-bladder, and could not have been overlooked at the time of the operation. It is highly probable that they were retained in some of the hepatic ducts and were washed into the bladder by the relief of pressure and free flow of biliary fluid, which came on after the formation of the fistula. Their presence seemingly had little to do with preventing the flow of bile into the intestine, at least the patulency of the common duct was not restored by their removal.

The remarkably profuse flow of muco-biliary fluid, coming through the sinus established, argues very strongly in favor of the operation, which contemplates the formation of a fistula in the gall-bladder in distension thereof by confined secretions. Certainly I think no tube of the dimensions of the ductus communis choledochus could have given it free exit; at least the accompanying back pressure on the walls of the cyst would have greatly endangered, if not certainly destroyed, the adhesions in any recent wound thereof, even when protected by the continued suture. Most of the cases of reported cholecystotomy which I have been able to consult, tell of the presence of a free flow through the fistula, so that it is a condition to be expected, and makes immediate closure of the bladder wound without drainage a dangerous proceeding to adopt, even if one can be positively sure of a clear common duct.

In a recent letter from Mr. W. T. Meredith, of London, in reply to some inquiries about his case of cholecystotomy, Mr. M. says a large amount of biliary matter was found in the gall-bladder. It was tapped with a trocar and then freely opened. Three large stones were found and removed, and as the common duct seemed entirely patulous, the opening in the bladder was carefully closed, no drainage adopted, and the external wound closed. The operation was done under the spray. The patient

died inside of forty-eight hours with suppression of urine. On post-mortem examination the gall-bladder contained one-half pint of bile and considerable quantities (several ounces) were found in the folds of the mesentery; some bile had also gotten into the duodenum. The gall-bladder incision seemed entirely closed—none of the fluid coming out on squeezing the cyst. The kidneys were found diseased, and their condition evidently mainly led to the fatal issue.

Still, Mr. Meredith says: "As regards the dispensing with all drainage, I am not disposed to repeat the procedure above described, although I do not see why it should not succeed in a favorable case, provided always that it is possible to ascertain with certainty that the ducts are left quite free. This, of course, constitutes the weak point in the matter, and, on the whole, therefore, I think the usual plan of drainage and suturing to the abdominal wall should be preferred."

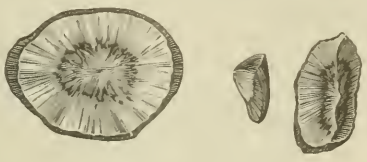
This is the only case of immediate closure that I have any account of in which the report is at all full. I am disposed to go further than Mr. Meredith, by expressing my convictions that the back pressure of accumulated secretions following a long period of occlusion must have an additional safety valve, and that the safety of the patient demands the establishment of a fistula when possible.

Naturally follows the question of whether it is always possible to establish a fistula in cases of gall-stone obstruction. Within a week of the time of doing the operation above reported, this question was practically answered by a post-mortem examination on the body of a lady who had suffered for some years with all the symptoms of gall-stones. The examination revealed the gall-bladder shrivelled to the size of the end of the thumb, and the common duct distended and containing six gall-stones of the size of the two now shown to you, which are of the six found in this case. In this case the bladder could not have been brought in contact with the abdominal walls, as the patient was not thin, and the intestines were full and of large calibre. It is quite certain an operation in this case would have presented many difficulties. Here incision of the duct, removal of the stones, and immediate

closure by means of the intestinal stitch would have been, perhaps, the only operation to do. In such a case to me the best practice would seem to require the insertion of a drainage-tube to the site of the duct wound, bringing it out of the abdominal opening, in order to provide for external exit, should any leakage occur.

Nature's methods of freeing the bladder and ducts from these foreign bodies are unique, interesting, and sometimes dangerous. Here, for instance, are shown to you several gall-stones which produced death by ulcerating their way into the stomach through the walls of the pylorus. They were found in the stomach after a post-mortem examination in a case under the care of Dr. C. T. Fenn, of Chicago, Illinois (Fig. 2).

Fig. 2.



Through the courtesy of Professor Hosmer A. Johnson, of Chicago, I am enabled to show this valuable specimen. It displays a very large gall-stone entirely filling the lumen of the small intestine. The patient suffered for several days from the ordinary symptoms of complete obstruction of the bowel, and finally died with all the usual signs thereof present. The subsequent autopsy not only gave this specimen but showed as well the large opening made by ulceration between the gall-bladder and duodenum, through which it passed into the bowel. A full account of this case was published by Dr. Johnson, July 1, 1875, in the *Chicago Medical Examiner*. Dr. Johnson therein also gives some valuable suggestions as to the procedures to be adopted for relief of the obstruction had a laparotomy been done.

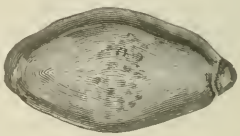
This beautiful specimen of pure cholesterine (weighing 174.29 grs.) was removed from an abscess of the abdominal walls opened near the anterior superior spine of the right ilium. The patient recovered entirely after a few months' trouble with a

fistula (Fig. 3). Finally this specimen was removed by myself a month or so ago from the centre of a solid tumor occupying

Fig. 3.

partly the abdominal walls and dipping into the abdominal cavity in the region of the gall-bladder. The operation was exploratory in character to determine the nature of the tumor and this stone (weighing 42.46 grs.) was accidentally hit upon (Fig. 4). Microscopical examination of sections of the tumor show

Fig. 4.



nothing but fibrous tissue. Was the tumor a result of the irritation incidental to presence of this stone? It had begun to disappear.

A reasonably sure diagnosis can be always made by means of exclusion. The chronic character of the symptoms, their prolonged existence without any especially noticeable changes in surrounding organs with accompanying systemic manifestations are characteristic.

The paroxysmal pain accompanied with slight jaundice, coinciding as it does with periodic biliary secretion and flow, is almost pathognomonic. The presence of a determinable tumor adds to the certainty. To my way of thinking exploratory puncture with the needle is neither safe, desirable, nor necessary.

The safety of the operation is plainly demonstrated by the

table of operations so far given. Ending September 3d, 1884, Drs. Keen and Musser tabulate thirty-five cases. There have been reported since then two cases by Mr. Tait, one by Mr. Savage, one by Mr. Taylor, and one by myself, bringing the total number of operations up to forty.

In this list are found recorded ten deaths, but no deaths are shown to have followed uncomplicated cases of cholecystotomy. The relief given seems so far to be almost immediate as well as permanent.

Dr. J. McF. Gaston, of Atlanta, Georgia, has published in the *Atlanta Medical and Surgical Journal*, March, 1885, and *The Southern Medical Record*, March, 1885, some very complete and commendable papers on the surgery of the gall-bladder, embracing, also, a very graphic *résumé* of the attending troubles requiring surgical interference in diseases of that organ. In these papers Dr. Gaston advocates the establishment of a fistula between the bladder and the duodenum, by fastening the walls of the two viscera together; subsequent ulceration of the stitches establishes an opening between the gall-bladder and duodenum, instead of making an external fistula, in this way providing a passage for the biliary secretions along their normal channel and securing all its benefits to the general system. He has successfully performed the operation recommended upon dogs.

Nature often takes the same course, and practically secures the same kind of a fistula as Dr. Gaston advises to be made in many attempts to get rid of these calculi. One objection to the adoption of such a procedure rests in the fact, that it enhances, perhaps greatly, the danger of an operation, which has so far been proven to be comparatively safe and entirely reliable in giving relief.

It would be presumptuous to advise the general practice of sounding the common duct through the external opening, from its results in this single trial; still it is quite sure that such procedure can be safely carried out, and equally certain that its adoption in this case resulted in relief that did not follow the removal of the calculi alone.

DISCUSSION.

Dr. J. Ewing Mears, of Philadelphia, Pennsylvania.

Mr. President, this paper is one among those read at this session, which I esteem of great interest, because, as I have taken occasion to say before, I think it is the work of this Association to take up each year the new questions presenting themselves in surgery, and by carefully prepared papers and carefully elicited discussion to send forth to the world the opinions of the Fellows of the Association upon these questions, and in this way contribute to the advance of surgical knowledge.

This subject presents two aspects in which it may be discussed. It is but a few years since the questions relating to hepatic colic, impacted gall-stones, and the discharge of gall-stones into the intestine were under the care of the physician. The surgeon was not called on to interfere, and therefore the subject has been studied more by the physician, than by the surgeon. The time has come when the surgeon should study this question as the physician does. The operator should be not only a good surgeon, but also a good physician if he takes charge of these cases alone. It is possible for the physician to call the surgeon in to do the operative work, but the question of diagnosis is so important, as preliminary to operative procedures, that the surgeon should be fully acquainted with the medical aspects of the subject.

Cholecystotomy proper is called for in conditions other than the one reported in the paper. There are certain intrinsic and certain extrinsic conditions that may demand it. Certain conditions occurring in connection with the gall-bladder and the gall-ducts call for operative interference. These obstructive conditions lead to very serious hepatic diseases; diseases which possibly may not be remedied in these cases. Therefore operative procedures are called for early, and this is a point which it is well to bear in mind.

The conditions which are regarded as calling for operative procedures are, first, inflammatory conditions in the interior of the duct affecting the lining membrane and producing obstruction; second, tumors pressing upon the duct and making pressure so as to cause obstruction; third, malignant disease of the tissues surrounding the duct and causing obstruction; fourth, a condition of

suppuration in the gall bladder, in other words, an abscess of the cyst requiring evacuation of its contents; fifth, the removal of gall-stones impacted in the duct proper, or in the common duct. The cases in which the operation has been performed and which have been reported, do not exceed fifty. In the cases reported by Drs. Keen and Musser, the list reached thirty-five cases with ten deaths. Gall-stones were not found in all these cases. The operation in some cases was done for other conditions. In two of the cases reported by these gentlemen no gall-stones were found.

We deal with the abdominal cavity to-day with so little fear that the suggestion has been made and discussed, as regards the propriety of exploratory operation in all cases, the ground being that taken by some in regard to the opening of the cranial cavity, that it is a simple procedure, and if it does no good it does not do much harm. The operation of laparotomy is attended with much danger, and we regard this operation, as we do trephining, a serious one, and we think it should not be done without due consideration.

It is incumbent on any one who attempts to treat these cases that he should be thoroughly informed as to the methods of diagnosis. I was glad to see the accurate report given in this case, because by a careful study of the symptoms we can make the diagnosis with less fear of error. We are all familiar with the ordinary symptoms of hepatic colic, but there are other conditions in connection with this operation which require very acute diagnosis.

I was interested in the portion of the paper which related to the method of operation, and to the point at which the abdominal cavity should be opened. In some cases which have been reported, laparotomy has been performed by making an incision in the median line. Then the incision has been made directly over the tumor, no matter where that may be. The question of hemorrhage occurring in the course of the operation has attracted attention, and this has some bearing upon the point of incision. In the linea alba the incision is apt to be attended with less hemorrhage than incision along the margin of the ribs.

There is one point which attracted my attention in this report, and that was the difficulty which Dr. Parkes experienced in getting at the gall-bladder. It seemed to me that the difficulty arose from the fact that the incision was made too high, and that it would always be desirable to make the incision so as to get on a line with the margin of the liver. If we have large cystic distension of the

gall-bladder, it can be reached through the abdominal wall if it comes in contact. If it exercises sufficient pressure to press up the liver, the first structure met with is the liver, which must be displaced in order that the cyst can be reached. It may happen sometimes that the inflammatory conditions which have taken place, are such as to make it manifestly impossible to displace the liver. Therefore it seems to me that in opening the abdomen in the performance of cholecystotomy, we should enter the abdominal cavity so as to get on a line with the lower border of the liver.

There is another point, and that is in reference to stitching the walls of the cyst to the abdominal wall; in other words, with regard to the formation of a fistula. This is one of the methods of operation which has been employed. In the first case of cholecystotomy, which was performed without premeditation by Dr. Bobbs, another method was used. He opened the abdominal cavity, not knowing the condition which he was to encounter. There was a cyst of some kind. He found a cyst very largely distended, which he opened and from which he removed gall-stones. He called the operation lithotomy of the gall-bladder. In closing the wound which he had made he sutured the edges of the cyst together, and closed up the abdomen, the result being successful. This has been done in a number of cases, and is, I think, the method of operation to be preferred. The objection is, of course, the danger of the escape of bile into the peritoneal cavity. The adhesion which takes place from the introduction of sutures may be so firm as to prevent the escape of bile. The fistula which is left by the method of operation employed in the case reported by Dr. Parkes is usually temporary. It is much more temporary in character than the fistula which occurs as the result of the spontaneous opening of an abscess of the gall-bladder. This form is apt to become permanent.

I desire to refer to one or two other points in regard to the treatment of the gall-bladder after laparotomy. The opening of the cyst and the removal of gall-stones is only of temporary benefit, unless the condition which produces the gall-stones, the cholelithiasis, as it is termed, is removed. The patient may again present the symptoms indicating the impaction of gall-stones. It has been suggested, in order to gain a permanent result, that the gall-bladder should be removed, and in that way the danger of recurrence prevented. That has been done by excision, and also the duct has been removed in certain cases, and the gall-bladder attached

to the duodenum at another point. With regard to the propriety of this operation I have nothing to say. We must consider the question whether or not we should remove the gall-bladder, even when disease is present. While we are disposed to regard the gall-bladder as performing a mechanical duty, in serving as a reservoir for the bile, it may be that in health there is a certain relation between the secretion by the liver and the accumulation of the bile in the gall-bladder, which is necessary to the preservation of the organ in a condition of health. If the bladder is removed this relation is destroyed. This is a subject which requires further study and clinical experience.

Dr. L. McLane Tiffany, of Baltimore, Maryland.

I have two or three things to say in reference to the paper. First as to the difficulty of diagnosis. How impossible it is for the most competent operator to say exactly what the condition of things is inside of the abdomen! Although the diagnosis was made here properly enough, yet the tumor coming under the surgeon's hand, and supposed to be the gall-bladder, proved to be the liver on top of it. This is another of the many illustrations of the impossibility, with our present knowledge, of our being able to make an absolutely positive diagnosis, and the importance of being prepared for something more or less different from what was expected.

Another point is in reference to the behavior of different organs when certain of their ducts are closed by a foreign body. Where organs are bilateral they are extremely intolerant of the presence of a stone. A kidney subjected to the presence of a stone for the length of time that this gall-bladder was would quite certainly have become disorganized. Here, on the other hand, is the liver, whose ducts have been interfered with for a number of years, and little or no bile finding its way into the alimentary canal. At the same time, although the patient was pasty looking, life was carried on and the patient was pretty well nourished. The behavior of the liver with its ducts obstructed is extremely interesting when compared with the kidney. Again the ducts of the pancreas have been occluded, and yet life goes on, although with more or less disturbance in certain functions, and with more or less pain, but death as a result of such obstruction is not frequent. It has been my fortune in one case to see a salivary gland plugged with multiple calculi. This was the submaxillary gland, and it became necessary to remove it. It was

incapable of carrying out its function. It contained probably fifteen or sixteen calculi. Had the liver behaved in this way the consequence would have been disastrous. Especially in the liver do we recognize that it can be dilated without serious inconvenience. In trouble with the right side of the heart, nothing is more common than to see the liver pulsating, and this is one of the diagnostic signs of certain diseases of the heart. Certainly it is extremely interesting, the differences which we find between organs which are bilateral and those which are unilateral.

Catheterization of the duct might properly be spoken of. It seems to be an extremely good procedure and followed with good results. The bile, which was absent from the bowel, makes its way into it, the condition is improved, and the patient is able to get along with the gall-bladder closed, but whether or not it will be sufficient to carry on life is a question for future settlement.

Dr. Theodore F. Prewitt, of St. Louis, Missouri.

There are a number of interesting points about this case. While I have never had the opportunity of doing cholecystotomy I have had two or three cases in which I wanted to do it. As Dr. Tiffany has well said, the first important point is the diagnosis, and it is not always easy, still we can very often find the biliary tumor. I recollect a case which I saw fifteen years ago. It was that of an old gentleman who had never had any symptoms of gall-stones. Had never had hepatic colic. He was then seventy years of age. He was taken suddenly with severe pain and intense collapse. I was called in consultation. It was evident that it was a case of hepatic colic, and, upon examination, we could trace the gall-bladder as distinctly as if it were external, and could determine that it was as full of gall-stones as a boy's purse is with marbles. He never recovered from the collapse. Nothing had any influence upon that condition. At the autopsy the correctness of the diagnosis was proven.

I recall another case, that of an old negro who had what we call dropsy of the gall bladder with jaundice. I could readily trace the outline of the gall-bladder, which certainly extended to the level of the umbilicus or considerably below it. Fluctuation could be readily determined. I aspirated and drew off a pint of this mucoid fluid with very little indication of bile. I think that I aspirated a

second time. Evidences of stones were also discovered. I wanted to operate, but the man would not consent.

It is clear that we may have gall-stones which may exist for years without serious trouble. The presence of stones in the gall-bladder does not necessarily give rise to as much irritation as is ordinarily supposed. In the first case the gall-stones must have been present for twenty-five or thirty years, and yet the patient never had a single symptom referable to gall-stones. The mere presence of gall-stones would not be sufficient to demand operation, unless symptoms of obstruction were present.

There are cases in which for years there are obscure symptoms which are not correctly interpreted. I recollect a case in St. Louis of a lady who had been an invalid for years, and was said to be hysterical, and doubtless was to a certain extent. A year ago I saw her in consultation for obstruction of the bowels. The diagnosis of impacted gall-stones had been made. When I saw her there was acute obstruction, and the symptoms were urgent. We laid the case before her, but she wanted to think it over. In a few hours she made up her mind to have the operation done, but then she was in collapse. At the post-mortem we found a large gall-stone which had made its way into the jejunum, and had followed the small bowel for probably twenty feet. It reached a point beyond which it could go no farther, and blocked the bowel and had produced the symptoms of obstruction. In this case, if the diagnosis had been made earlier and the operation performed, life might have been saved.

There are cases, perhaps, where the operation could be done, where there are no gall-stones, but where symptoms referable to the gall-bladder are present. The case of Dr. Hodgen, of St. Louis, was possibly of this kind. He died of perforation of the gall-bladder from a minute ulcer. For years he had been subject to pain about the region of the gall-bladder. On the morning of his death I had seen him, and he had remarked that "he had one of his old attacks of belly-ache." An hour or two later he was seized with a violent pain and excessive suffering, and died within about thirty-six hours. A minute opening was found in the gall-bladder permitting extravasation of bile. It is probable that gall-stones had existed for some time, resulting in the formation of an ulcer which had finally caused perforation.

Dr. Mears has alluded to the point of incision. It seems to me

that if you make the incision low down you will have a strain on the gall-bladder when united to the walls. The bladder is ordinarily below the margin of the ribs. It is only in cases where the liver is distended from the damming back of the secretions that you find it projecting. When you have given exit to the fluid the liver will subside, and if you make an opening low down and attach the walls of the cyst to it, you will have increased dragging on the attached walls. I think that the operation as done was proper, for with the shrinking of the liver there was less contraction.

In regard to cholecystotomy this case points out the danger to be considered in cases of that kind. Here, after the opening had been present for some time, and after drainage had taken place, still, when the opening was closed, the patient suffered pain. That may be said to have been due to accumulation in the gall-bladder. It resulted from narrowing of the common duct by previous inflammatory conditions. Even if the gall-bladder had been removed, it could not have accommodated itself to the increased flow of bile, and the same condition would have resulted. It seems to me that this is a decided objection to cholecystectomy. When the gall-bladder is left drainage is an important feature, because the cyst-wall is almost always in a diseased condition. The distension of the gall-bladder is not due to accumulation of bile. In the cases which I have seen there has been a mucoid fluid from a sort of cystitis of the walls of the gall-bladder. Drainage is a very important feature, and I think it advisable to keep the opening patulous.

There is one point in regard to the gravity of the case, where we have fistula and where we have no fistula. Where an abscess forms and a fistula is present it is a simple matter. I recollect a case of abscess of the gall-bladder in which I aspirated the abscess. A fistula formed, gall-stones were removed, and the patient got well. There union had taken place by inflammation; but opening of the abdominal cavity is not entirely a harmless operation.

Dr. S. W. Gross, of Philadelphia, Pennsylvania.

I have collated 42 cases of operations upon the gall-bladder for concretions or dropsy, an examination of which may prove of interest in our efforts to arrive at correct conclusions regarding the proper management of these conditions.

Of 36 cases of cholecystotomy, 28 recovered, and 8, or 22.22 per cent., died—3 of exhaustion and hemorrhage, 2 of shock, and

1 each of escape of bile into the peritoneal cavity, suppression of urine, and acute peritonitis. The operators were Tait in 15 cases, Keen in 2 cases, and Kocher, Ransohoff, Gardner, Eddowes, Savage, Bobbs, Sims, a surgeon quoted by Tait, Courvoisier, Trendelenburg, Koenig, Boeckel, Naire, McGill, Meredith, Parkes, Savage, Taylor, and Wright, each in 1 case. In the case of Wright, the patient was thought to be the subject of renal calculus, so that the incision was made through the loin, and a stone extracted from what at first was presumed to be the enlarged ureter. On death from peritonitis the kidney was found to contain a stone. In 4 cases the gall-bladder was sutured and returned into the belly. Of these 1, or 25 per cent., died as the direct result of the procedure, as against 8 deaths out of 31 cases in which the viscera was stitched to the wound, the mortality being 22.58 per cent. In the case of the surgeon mentioned by Tait, the sutures appear to have given way and permitted the escape of bile into the peritoneal cavity. That suturing the bladder is not so dangerous as Tait assumes is shown, not only by the comparatively insignificant rate of death, but by the case of Meredith, in which, on death from suppression of urine, the stitches were found to be secure, and the gall-bladder contained nearly eight ounces of bile. Were I myself called upon to do the operation, I would certainly employ sutures and return the organ.

In 6 cases the gall-bladder has been excised, 5 times by Langenbuch, and once by Courvoisier. One of Langenbuch's patients died, because of the existence of an ulcer in the cystic duct, which was overlooked on account of the presence of large masses of fat, which permitted the bile to escape into the peritoneal cavity. Hence, the mortality of cholecystectomy is 16.66 per cent., or 6.19 per cent. less than that of cholecystotomy, which shows that it is not so absurd a procedure as Tait declares it to be. Finally, in 1 case Von Winiwarter successfully established a communication between the duodenum and the gall-bladder. This operation, or cholecystenterostomy, has not been repeated on the human subject, although Dr. Gaston, of Atlanta, has experimented with it on dogs.

In the present state of our knowledge, based upon the foregoing data, I would prefer cholecystotomy in recent cases, but would not hesitate to extirpate the gall-bladder if its coats were ulcerated, or if ulceration were threatened.

PHOSPHORUS–NECROSIS OF THE JAWS.

By J. EWING MEARS, M.D.,

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THE earliest observations made with regard to the occurrence of phosphorus-necrosis of the jaws were those of Lorinser, of Vienna, who published a paper upon the subject in 1845, recording cases which occurred as early as 1839; Heyfelder, of Erlangen, and Strohl, of Strasburg, shortly after reported cases and confirmed the observations made by Lorinser. In 1846 Dr. Wilks, of London, published a paper upon the subject in *Guy's Hospital Reports*. In 1847 an exhaustive paper was published by Professors Von Bibra and Geist, of Nuremburg, which will be alluded to hereafter. In 1850 Mr. John Simon, F.R.S., delivered a clinical lecture at St. Thomas's Hospital, London, on diseases of the lower jaw produced by phosphorus fumes, illustrated by the report of two cases at that time in the Hospital. Other cases were reported, by both English and Continental surgeons, between this period and 1862–5.

Soon after the report of the cases occurring in Germany the attention of the authorities was attracted to the subject, and stringent regulations were made with regard to the adoption of preventative measures in the match manufactories of that country. In 1863 Mr. Simon made a report to the Privy Council of England with regard to the prevention of the disease. His report contained the results of the careful investigations made by Dr. Bristowe upon the subject. In 1867 Mr. Christopher Heath, in the Jacksonian Prize Essay of the Royal College of Surgeons of England, gave a full account of what was known upon the subject up to that date. In this country Dr. James R. Wood, of New York, was the first to give an account of the

disease, accompanied by the history of a case in which he removed the entire inferior maxilla. Subsequently cases were reported in which the entire lower jaw, or portions of it, were removed by Drs. Wm. Hunt and C. S. Boker, of Philadelphia, and one notable case by Dr. J. C. Huchison, of Brooklyn, in which the entire upper jaw, with the malar bone, was excised.

The text-books on surgery contain more or less full accounts of the disease; those of Profs. Gross and Agnew treat the subject at some length.

In order that information may be imparted with regard to the manner in which necrosis of the jaws is developed in connection with the manufacture of matches, it may be of interest to detail briefly the process of manufacture. The essential parts of a match are the stick, the purpose of which is to hold the flame for a sufficient length of time, and the chemical composition which is fixed upon the end, and which, by friction, ignites the stick. The stick, in order that it may take fire readily, is charged, in the common sulphur match, with sulphur, and in the finer quality of parlor match, with paraffin. The chemical composition, which tips the stick, and by friction ignites it, consists in the finer quality of match of phosphorus and chlorate of potassium, inflammable materials, with particles of ground flint to assist friction, a coloring agent, and the best quality of Irish glue to give the mixture body and adhesive properties. The tipping of the stick by the chemical composition is accomplished by dipping the ends in a warm solution of the composition, placed in shallow pans, and maintained at the proper temperature by a steam-bath. From these dipping pans fumes constantly rise into the faces of the workmen or dippers. Both ends of the match sticks are dipped, and after being allowed to dry sufficiently to permit handling, which is accomplished in four to six hours, the sticks are passed through the cutting machines and divided. In passing through the cutting machines the friction is often sufficient to cause ignition of large bundles of matches, the fumes from which envelop the cutters and keep them almost constantly surrounded by the vapor from burning tips. From the cutting machines the matches go to the pack-

ing-rooms, where they are assorted and packed, this process requiring the handling of the tips, not yet thoroughly dry, and from which vapor is constantly emitted. In addition, ignition of the matches frequently occurs, filling the packing-rooms with the fumes from the burning matches, and which the employés inhale. The stages of the process are, therefore, charging the stick with sulphur or paraffin, dipping the ends into the chemical composition, cutting the dipped sticks, and finally packing in boxes. No danger manifestly attends the process in the first stage, as neither sulphur nor paraffin is capable of producing the effects observed in necrosis of the jaws as it occurs in the employés in match factories. In the dipping process the fumes arising from the composition in heated solution are inhaled by the employés, while the material adheres more or less to the hands in the manipulation which is necessary. In the cutting of the sticks handling of the moist tipped ends occurs, with the inhalation of the fumes constantly emitted from them and from the masses of matches continuously ignited by friction. The packing requires the constant handling of the matches, and, as a result, the hands of the employés are coated with the composition so as to appear luminous in the dark, and the ever-recurring ignition of bundles of matches fills the rooms with the vapor, enveloping the work-people in an atmosphere charged with the burning composition.

In match factories the employés consist largely of females, the work being of such light character as to require but little physical strength. The ages vary from twelve to twenty, the former being the time of life at which they usually enter the service. Men are employed to work the dipping machines, and a few boys are usually employed in general work.

The effect of the nature of the work upon the employés varies in different individuals. Some engaging service are compelled to abandon work in a few days, others in a few weeks or months, and again some continue in the work for years; one notable instance is that of a woman coming under my care who had worked for twenty-six years in a factory, and was at the expiration of that time attacked with necrosis. In many there is observed a gradual deterioration of physical condition as manifested in loss of

flesh and general vigor. Necrosis of the jaws occurs in those who have been employed for a period of years in the factories, usually not until four to six years have elapsed.

The physiological effects of the phosphorus vapor are stated by authors to be irritation of the mucous membrane of the eye and of the air-passages, while in medicinal doses, taken into the stomach, the action of the heart is increased, the temperature is somewhat elevated, mental activity and muscular power are augmented, aphrodisiac effects are experienced, the urine and perspiration are freely excreted, and, in females, the menstrual flow is increased.

In toxic doses violent gastro-intestinal disturbances are caused, the mucous membrane being attacked with great energy. Severe epigastric pain, with vomiting and diarrhœa, supervenes, and in addition to the physiological effect of the agent there is also evidence of the action of a corrosive poison. Phosphorus taken into the stomach passes into the blood unchanged, and if in sufficient quantity causes rapid destruction of the red blood-globules, prevents the reaction of the fibrinogen on the fibrino-plastic substance, and thus originates an acute hemorrhagic diathesis. Transudation of blood takes place from all of the mucous surfaces. According to Wegner, the hemorrhages are due to fatty degeneration of the arterial walls. This degeneration, according to other observers, extends to the stomach, liver, heart, and later to the voluntary and involuntary muscular systems.

I have had the opportunity of examining a large number of employés in match factories and of studying the effects of phosphorus upon them. In sixteen cases I have treated the toxic effects of phosphorus as it appeared in different stages of development upon the system, and I have been interested in observing to what extent toxic symptoms could be traced and defined. Of these cases the majority has been among those who are engaged in the packing room, where the handling of the matches occurs; but one was a dipper and four were cutters.

Of the symptoms described as giving evidence of physiological or toxicological effects, very few have been noted as prominent in the cases under my observation. Beyond instances

of conjunctivitis and bronchial irritation, I have not been able to elicit definite information as to any marked effect upon the membrane of the general mucous tracts. Constipation rather than diarrhœa has been the condition which existed. Examination of both male and female employés has not revealed any history of aphrodisiac effects, and in the latter, increase in the menstrual flow has not been noted to any extent. The most marked toxic symptom observed has been the occurrence of hemorrhagic transudations from the gums, and this symptom is frequently noted among the employés. Pain over the region of the liver, sick headache, nausea, and muscular debility, are symptoms described by many, while well-defined icterus has not been observed in any case. I have been informed of three fatal cases in which the immediate cause of death was phthisis, occurring some time subsequent to operations for the relief of necrosis of the jaw.

It is evident from the absence of symptoms of acute poisoning that the invasion of the system by the poison is slow, establishing a chronic toxic condition which awaits some exciting cause to make itself manifest. Accepting as parts of the pathological processes which follow toxic effects, disintegration of the red blood-corpuscles, and fatty degeneration of the arterial walls, we find in the highly vascular periosteum a membrane most liable to exhibit the effects of these morbid changes. Owing to the weakened condition of the arterial walls consequent upon the state of fatty degeneration present, the vascular tension is wholly or in great part lost, and the tissue, while surcharged with blood, is in a state of innutrition, owing to the impoverished condition of the blood—so that I have observed in cases free hemorrhages taking place from gums blanched and apparently bloodless. This condition is most favorable for the development of inflammation, and this process follows promptly upon the appearance of the exciting cause.

In 1847 Professors Von Bibra and Geist, of Nuremberg, published a valuable monograph upon the subject of phosphorus-necrosis, containing a very complete and clear summary of all that had been written previously upon the subject, and also the

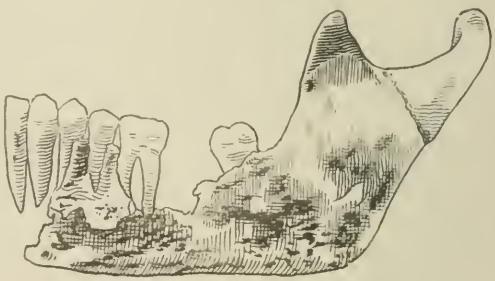
results of their very careful investigations. The conclusions arrived at were, among others, that phosphorus was the poisonous agent, and that the effects were entirely local. These statements were regarded as proven by the experiments upon rabbits submitted to the influence of phosphorus vapor. In these animals the characteristic disease could only be produced, according to the report, after laceration of the periosteum of the jaw and exposure to the vapor. As the result of these experiments the deduction was made that the agent acts only when it can come in direct contact with the periosteum, and that those employés in match manufactories who are free from carious teeth can pursue the occupation with impunity. They suffer neither locally nor constitutionally. The vapor appears to be incapable of producing any general cachexia.

While the observations which I have made agree, in general, with the conclusions stated above, it is my belief that the statement made with regard to the local effects and the introduction of the poison through carious teeth should be received with some modification. I have examined a number of employés who have had carious teeth and were yet free from the disease, and have continued to work in the factories exposed to the phosphorus vapor; also those who have had teeth extracted and have returned immediately to work in the factory and have not suffered from necrosis. A number who have suffered from the disease have given, as one of the symptoms which first attracted their attention and caused alarm, the sensation of a protrusion of a number of the sound teeth, or, as expressed by them, an elongation of their teeth—this condition would occur, and then disappear, being the result apparently of an accession of cold. In one case the teeth, on two occasions, became quite loose and the gums much swollen and very sensitive. This condition of protrusion of the teeth from the alveoli indicated an inflammation of the peri-dental membrane, a structure which is continuous with the periosteum, and which lines the walls of the alveoli and surrounds the root of the teeth. In all of the patients whom I have been able to examine in the early stages of the disease, I have found an accumulation of tartar around

the necks of the teeth; in some this tartar has existed in large plates extending beneath the gum and covering the bodies of several teeth to the crown. This accumulation is a source of constant irritation, and being lodged at the point at which the periosteum turns into the alveoli, gives rise readily to irritation of the peri-alveolar and peri-dental membrane; assuming the correctness of the statement as to the pathological condition which the periosteum acquires as a result of a chronic toxic condition, we find in the carious tooth or in the accumulation of tartar a source of irritation which is competent, under favorable conditions, to develop inflammation. It is not my belief that the poison impresses the membrane *primarily* through the carious tooth, as stated by Profs. Von Bibra and Giest, or the peri-dental membrane laid bare by the accumulation of tartar around the neck of the teeth which may have caused a recession of the mucous membrane and slight absorption of the alveolar process, but that these conditions, *per se*, under the influence of some exciting cause, as cold, etc., light up in the periosteum, already in a morbid condition, produced by constitutional impression of the poison, an inflammation, which, if not arrested, results in necrosis. The pre-existing condition, it seems to me, is therefore a morbid state of the periosteum, which under an exciting cause readily inflames; the exciting causes are found in the teeth; a carious tooth, with the pulp exposed, which structure, by some of its tissues, is continuous with the peri-dental and peri-alveolar membrane, and tartar accumulations may give rise to an irritated and sensitive condition of the borders of the reflected periosteum. It is a matter of observation that the periostitis which develops in connection with other causes, with the exception, possibly, of syphilis and mercurial poisoning, is, in many cases, of a local character, whereas that which precedes prosphorus-necrosis is, as a rule, general, passing rapidly to involvement of the entire bone, unless interfered with. In this, as in some other respects, it resembles syphilitic periostitis. In the tertiary form of syphilis the periosteum of the jaws is very prone to inflame under the irritation of carious teeth or the violence inflicted in the efforts at extraction. In the cases of phosphorus-necrosis coming under

my care, in the advanced stages of the disease, the uniform statement by the patient has been to the effect that, suffering pain in a carious tooth or in a sound tooth, a visit was made to the dentist and extraction was performed. In a day or two the jaw began to swell, and following that a discharge appeared from the point of extraction, the history of the beginning of suppurative periostitis caused by the efforts at extraction. In some cases I have found portions of the teeth remaining and the alveolar process greatly broken. The results of the experiments obtained by Profs. Von Bibra and Giest upon rabbits are not at variance with the views expressed above, since, according to the statements made, the animals in some instances were exposed for a period of time to the fumes of phosphorus before the periosteum was removed and the bone denuded, and it is quite reasonable to believe that being easily impressed by the poison, a morbid condition of the periosteum had supervened so that the laceration of the membrane was sufficient to excite the inflammation. In those animals in which the lower jaw was fractured, laying bare the roots of the molar teeth previous to the exposure to phosphorus vapor, there were evidences, on post-mortem examination, of serious constitutional, as well as local, effects.

Fig. 1.



Condyloid process covered with new bone formation.

The specific inflammation excited in the periosteum progresses rapidly, and results, in cases of the lower jaw, in the formation of laminated layers of new bone, which envelop the jaw, stopping at the borders of the alveolar process in front and

behind, but completely surrounding the angle, ramus, and both processes.

In a number of the specimens removed by me the new bone formation had encroached upon the articulating surface of the condyloid process (Figs. 1 and 2), and in one instance completely

Fig. 2.

Condyloid process covered with new bone formation.

covered both condyloid and coronoid processes (Fig. 3). An interesting result has occurred in one case under my observa-

Fig. 3.

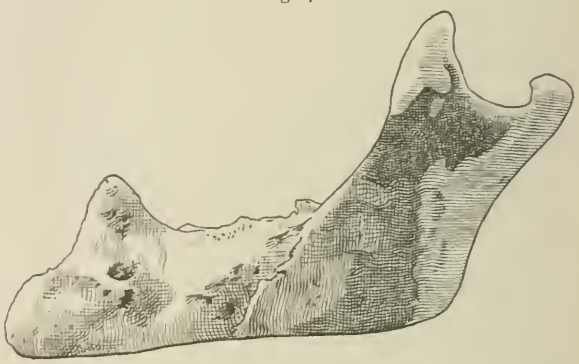
Condyloid and coronoid processes covered with new bone formation.

tion in which the patient refused operative interference; the necrotic action had continued for six years, and the layers of new bone had united over the position of the alveolar border,

thus entirely inclosing the sequestrum, which is slowly disappearing, the discharge escaping externally by a sinus at the angle of the jaw. The muscles attach themselves to the periosteum covering the new bone, and are lifted off, as it were, with that membrane.

In some of the specimens in my collection portions of the new bone have exhibited the pumice-like appearance described as characteristic of this specific form of inflammation (Fig. 4); this

Fig. 4.



Pumice-like deposit on body and part of ramus.

appearance has also been observed upon the surface of the sequestrum. In other instances it has been absent, in this respect confirming the opinion which has been expressed that it is not present in every case, and therefore not pathognomonic.

Profs. Von Bibra and Geist have made careful examinations microscopically and chemically of the bone deposited by the periosteum in these cases. It is described "as lamellated, with large Haversian canals, not running *parallel* to the general direction of the bone, but placed at right angles—the lacunæ are rounded off; the proportions of organic and inorganic matter vary, the average being in eight specimens—organic matter 32.4, inorganic 6.76."

Mr. John Adams, in a clinical lecture, published in the *Medical Times and Gazette*, July 5, 1852, states that these conditions are not observed in bone formed as the result of common inflammation.

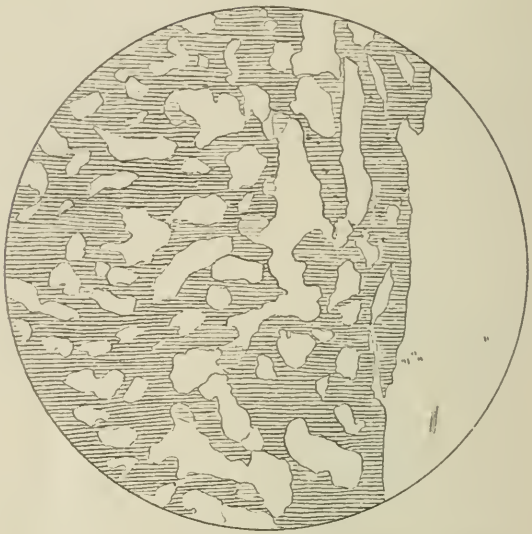
Mr. Thomas Smith, in the *St. Bartholomew's Hospital Reports*, vol. i., reports a case in which he removed the sequestrum of the entire lower jaw, the newly formed jaw showing on microscopic examination "all degrees of development, from a finely fibro-nuclear matrix up to perfect bone. The bone differs from ordinary compact bone in being excessively vascular, the Haversian canals being very large, near together, freely anastomosing, and here and there in their walls presenting fusiform and pouch-like dilatations, in fact resembling, in their outlines, veins slightly varicose. The bone is thickly studded with lacunæ, and these are peculiar in being very large in their cavities, less uniform in their general outline, and containing fewer canaliculi than is usual in well-formed bone. In the newest parts of the bone the lacunæ are merely irregularly-formed cavities without distinct canaliculi." These appearances correspond largely with those observed in general in new-bone formations, and do not seem to me to be sufficiently distinctive to confine their description to formations occurring as the result of phosphorus-necrosis.

I append the report of Dr. Morris Longstreth upon a specimen of new bone formation taken from one of the cases under my care, and submitted to him for examination.

In the specimen of periosteal outgrowth from the case of phosphorus-necrosis of the lower jaw, two sets of sections have been made; one, vertically to the surface, including the entire thickness of the growth; the other horizontally. The sections (Figs. 5 and 6) show as their striking features the enormous size of the canalization system and the slenderness of the bone-trabeculæ. In the horizontal sections the canals appear of a greater diameter than in the vertical sections. The thickness of the bone-trabeculæ appears about the same in whatever direction the tissue is cut. The inference to be drawn from this arrangement is that the canals, being connected with the vascular system, either of the jaw bone, on the one hand, or, on the other, with that of the periosteum, have their extension chiefly in the vertical axis. This mode of distribution is evident from a naked-eye examination of the specimen, and is especially evident on its under surface; the upper surface is denser and more compact. It would give a wrong impression to assert

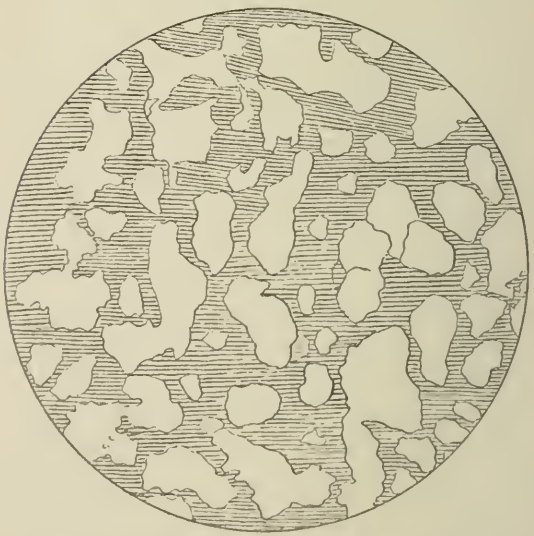
that the canalization is wholly in the direction of the vertical axis. The ramification of the canals proceeds in every direction through

Fig. 5.



Vertical section of periosteal outgrowth from phosphorus-necrosis of jaw.[1]

Fig. 6.



Horizontal section of periosteal outgrowth from phosphorus-necrosis of jaw.

The wood-cuts are copied from photo-micrographs taken with a ¼ in. lens; natural size about 50 ×.

the substance of the new formation of bone, as can be seen by the varying angles of obliquity at which the canals are found to be cut in the section. The new substance is rather like a sponge, in which the channels of the largest size are, in general, vertical, as is but natural, since the other channels with which they communicate are seated in the directly subjacent jaw, or the superimposed periosteum.

Compared with other periosteal outgrowths, originating from other causes than phosphorus-necrosis, it will be seen that the same arrangement of canalization holds true as in this specimen. Here, in vertical and horizontal sections from a periosteal outgrowth from a femur (Figs. 7 and 8), the horizontal sections show larger sized

Fig. 7.

Vertical section from periostitis of femur.

canals than the vertical sections, while the bone-trabeculæ in these two sets of sections (periostitis of femur) have about an equal thickness. The outgrowth from the femur shows about the same macroscopic appearances as that from phosphorus-necrosis, except that the former is much more dense; the latter may be cut with a knife, the former requires a saw for its division.

Compared with normal bone the chief direction of the canalization system is found to be governed by the same principles in the normal, as the pathological, growth. The law is that the canals

pursue the same general course as the blood supply most nearly contiguous to them. On or near the periosteal surface the canalization is more or less vertical, perhaps tending somewhat horizontally, inclined towards the distal extremity in the long bones; in the

Fig. 8.

Horizontal section of an outgrowth from periostitis of the femur.

deeper layers of the compact tissue of the long bones the canals conform in their direction to the long axis of the bone. In flat bones the canals are almost wholly placed vertically in reference to the periosteal blood-supply. In the periosteal outgrowth the canalization system is found, therefore, to conform to the law governing normal tissue.

In respect to the cellular elements, bone-corpuscles, etc., found in this periosteal outgrowth from phosphorus-necrosis, it is impossible to speak, since the specimen is a dried one from which the softer tissues have disappeared.

The lamination which is visible in the specimen, and which is of not uncommon occurrence in periosteal outgrowths, is much more apparent than real. A section made through the junction of two laminæ, very distinct to the naked eye, does not show to microscopic vision any differentiation of tissue, or any disturbance of the arrangement of the canalization system, which is apparently continu-

ous through the two superimposed laminæ. The laminæ are to be regarded as marking the steps of the growth—a temporary cessation of the formation of the periosteal deposit or outgrowth. The new lamina cannot be regarded as a *de facto* commencement of a new process, but merely as a continuation of the old morbid process, with a very short interval or halt.

With regard to the reproduction of bone after phosphorus-necrosis, involving the upper jaw, the cases under my care have confirmed the experience of others who have had opportunities to observe similar cases. In no instance has there been more than a development of fibrous tissue after loss of substance. M. Ollier, of Lyons, records one case of phosphorus-necrosis of the upper jaw, in which a certain amount of new bone was produced. He also quotes a case reported by Billroth, of a similar character. Mr. Heath, in his work on the jaws, states as his opinion that these cases must be regarded as quite exceptional.

The permanent character of the new bone formation has been called into question by Mr. Salter, who states in his article in *Holmes's Surgery*, vol. iv., that the "repair of the lower jaw is but temporary, for after a time, often a considerable time, the new bone diminishes by absorption to a mere arch." He has found this state of the bone in one case in which the lower jaw had been removed ten years, and asks as a question of theoretical interest and of practical importance how far this loss can be prevented by supplying it with a function through the means of artificial teeth. While more or less absorption does undoubtedly occur, it has been my observation that this condition is affected largely by the period of time in the deposit of the new bone at which, and the manner in which, the operation for removal of the sequestrum is performed. If removal of the sequestrum is delayed until well-formed plates of bone have been deposited and care is taken in separating them from the sequestrum to avoid their fracture or dislocation, the absorption which naturally follows will not seriously impair the permanent character of the new-formed bone (Fig. 9). In one case in which I operated in 1881, removing two-thirds of the lower jaw, from the articula-

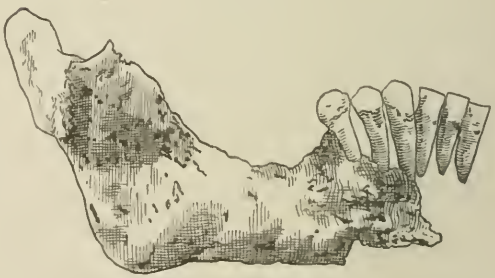
tion on the right side to the first molar on the left, the new bone formation has not exhibited any marked decrease in size up to

Fig. 9.

Appearance of patient after removal of right half of lower jaw—new bone well formed.

this period and the patient has been able to wear with comfort and satisfaction an artificial denture for the past three years. (Fig. 12.)

Fig. 10.

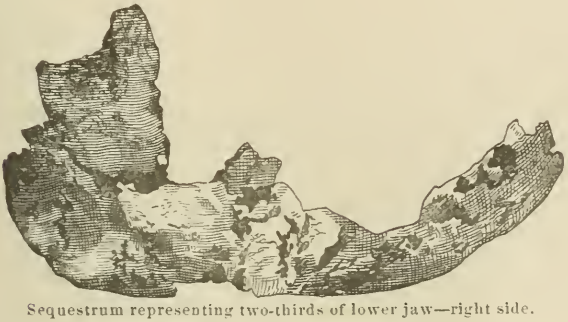


Sequestrum removed from patient shown in Fig. 9.

In another case in which a large portion of the new bone deposit had been removed as a primary operation, and with the intention of interfering in this manner with the progress of the dis-

ease, another layer of bone formed from the periosteal surface, which, however, lacked the density and firmness of the lamellæ

Fig 11.



Sequestrum representing two-thirds of lower jaw—right side.
(From patient shown in Fig. 12.)

Fig. 12.

Appearance of patient with artificial denture after removal of two-thirds of lower jaw—from right side.

deposited originally; as a result a greater degree of deformity occurred in the case than in others in which care was employed

to remove the sequestrum without disturbing to any extent the new bone formation. Whether the adaptation of artificial dentures will in any degree diminish the tendency to absorption is to me a question; it would seem that the pressure exerted and friction produced by the movement of the denture upon the newly formed and rather imperfect bone would develop absorptive action rather than diminish it. Notwithstanding, I always endeavor to have dentures adapted in order to supply the loss so much felt by patients.

With regard to the relative frequency with which the upper and lower jaws are attacked, the cases under my care have shown a decided proportion in favor of the lower jaw. Out of sixteen cases the lower jaw was involved in eleven, and the upper in five; in three both upper and lower jaws were attacked, the upper in a much less degree than the lower. This preponderance in favor of the lower jaw has been observed by the German and other authorities. Of fifty-two cases reported by the former, as stated by Mr. Heath, twenty-one were of the superior maxilla, twenty-five of the inferior, and five in which both were involved. Mr. Salter, *Holmes's Surgery*, vol. iv., records five cases under his care, in four of which the lower jaw was diseased, and the upper in one. In seventeen instances of which he obtained information, nine were connected with the superior and eight with the inferior maxilla. In Agnew's analysis of 111 cases, 58 were of the lower and 41 of the upper, with 12 in which both were involved. From the information I have been able to obtain with regard to cases occurring in this country, the lower maxilla appears to have been attacked more frequently than the upper. These cases show, as in those under my care, that the upper jaw is not, as a rule, involved to the same extent as the lower—the disease being limited rather to the alveolar border. This comparative immunity enjoyed by the superior maxilla is to be explained, it seems to me, by the difference in the configuration, the character of the structure, and the blood supply of the two bones. The presence of the sinus within the body of the superior maxilla lined by a membrane endowed with a good blood supply would enable the bone to resist the advance of the

necrotic action. The absence of a large amount of cancellous tissue as compared with the lower jaw would also favor this immunity, although Mr. Heath is of the opinion that necrosis occurs less frequently in cancellous than in compact bone. Such has not been my observation in connection with necrosis of the lower jaw arising from various causes, since in many instances the dense compact base of the bone seemed as it were to afford a barrier to the advance of the disease.

The *symptoms* of phosphorus-necrosis differ from those of ordinary necrosis mainly in intensity. The pain, which is one of the earliest symptoms, is at first intermittent, but in time becomes continuous. It is also more diffuse from the first, involving the entire bone. The first and second stages are, as a rule, rapidly passed through, and suppurative periostitis manifests itself in the peri-alveolar and peri-dental membranes causing the teeth to become loose and pus to exude from the alveoli. The inflammation quickly extends, involving the structures of

Fig. 13.

Necrosis of left half of lower jaw with sinuses opening at the base.

the gums and detaching them so as to expose the alveolar border. Infiltration of the tissues of the face follows, and swelling, which produces marked deformity, occurs. Soon the in-

flammation invades the entire periosteal layer, and the formation of the laminated bone can be distinguished, and offensive discharges escape into the mouth; in the case of the lower jaw the pus gravitates to the base and escapes by sinuses at this point (Fig. 13) or at some point lower down. In one case under my care the sinus had opened at the upper border of the clavicle before the patient presented herself to me for treatment. In some instances the pain continues during the suppurative action —as a rule, it is much diminished or disappears. When the inflammation involves the condyle of the lower jaw, great pain is experienced in the ear of the affected side, and occlusion of the jaws, which before may have been partial, now becomes complete. The interference with the ingestion of proper food owing to the condition of the mouth, the suffering endured by the patient associated with the constant presence in the mouth and the swallowing of the offensive discharges from the dead bone, all combine to deteriorate in a marked degree the general health of the patient. Unless proper remedial measures are resorted to, death may ensue from exhaustion, or in very severe cases, from gangrene of the overlying soft structures. In rare instances, such as the one alluded to in another part of this paper, the disease may assume a chronic form and the general health may be maintained during a period of years, whilst the sequestrum slowly disintegrates and the débris is carried off through sinuses.

The *treatment* may be divided into that of three stages, primary, intermediate, and secondary. The primary stage may be described as that in which the periosteum, in its morbid condition, manifests the results of beginning irritation. Unless alarmed by the occurrence of necrosis in a fellow workman, the patient rarely presents himself for treatment in this stage. Pain in the bone of a diffused character—intermittent in occurrence, with the sensation of protrusion of the teeth—soreness in the gums, with hemorrhagic transudations during mastication or upon the use of the tooth-pick, are the chief symptoms present in this stage. An examination may reveal one or more of the teeth in a carious condition; around the necks of the diseased, as

well as of the sound teeth, there may be found a collection of tartar extending some distance beneath the gums and displacing the soft tissues, while a softened condition of these structures with a discoloration of a purplish hue may be present. In this stage the treatment relates to the condition of the teeth, which should be conducted with the utmost care in order to avoid the production of periostitis, and the administration of remedies which are antidotal in character. Those teeth which are too much diseased to be saved by treatment should be extracted, especial care being taken to avoid fracture of the tooth or of the process. Collections of tartar should be thoroughly and carefully removed, the instrument being carried well up under the gums to remove any that may be lodged in this position. The carious teeth should receive careful treatment so as to relieve the sensitive condition which exists, and to avoid by gentle manipulation the development of peri-dental inflamnation. Temporary fillings should be inserted until such time as it may be prudent to employ those of more permanent character. The soft structures should be bathed with detergent and astringent lotions, as the phenol sodique, alcohol, tincture of myrrh, etc., in dilute solutions.

The constitutional treatment should consist in the administration of anodyne remedies to relieve pain and soothe the general system, and of that agent which is regarded as possessed of the power to neutralize the effects of the poison and arrest the disease. Dr. Letheby, in his work on "Chemistry of the Poisons," recorded his observations upon the power of the vapor of turpentine to neutralize that of phosphorus. Andant, quoted by Bartholow, was the first to report cases in which the antidotal properties of turpentine were observed, and Personne, by experimental demonstration confirmed the observation of Andant. The latter further inferred that "as turpentine destroyed the luminosity in the dark and arrested the escape of the vapor of phosphorus, that it acted similarly as an antidote in preventing the combustion of phosphorus in the blood and the consequent consumption of the oxygen." Höhler and Schimpf (Bartholow) showed by experiment that the commercial oil of turpen-

tine, that which had been exposed for some time to the air, and was, therefore, rich in ozone, was the only variety efficient as an antidote, that this form prevented fatty degeneration of the tissues, and that under its influence no free phosphorus could be found in the animals experimented on.

In the cases which have come under my care, five have presented themselves in the first stage of the disease, and in these I have been enabled to employ the plan of treatment outlined above. In two treated some three years since, the disease was arrested in the primary stage. At present I have three patients under this plan, and in these there are positive evidences of improvement.

In the stage which I have designated as the intermediate, in which the inflammation of the periosteum is well established, the plan of treatment should be such as to arrest, if possible, the inflammatory action and thus limit the destruction of bone. Active antiphlogistic measures are here indicated, free incisions of the periosteum, the knife being carried to the bone, should be employed, and the use of such internal remedies as will assist in combating the condition of inflammation. According to my experience the efforts to arrest the progress of the disease in this stage are, as a rule, without much avail; the specific character of the inflammation, so far as relates to its cause, and the rapidity with which it becomes general, seem to resist any successful interference. Measures should always be adopted, however, with the hope of at least limiting the subsequent necrosis.

When the inflammation has passed to the suppurative stage and the death of the bone accomplished, the treatment should consist in that which is supportive and expectant. Warm water should be freely used to cleanse the mouth, after which very dilute solutions of carbolic acid, iodine, or permanganate of potassium, should be thrown upon and around the exposed bone with a syringe so as to disinfect the parts and remove the collections of pus. A layer of 7½ per cent. iodoform gauze should be packed around the exposed bone so as to receive the secretions and prevent as far as possible their escape into the mouth and mixture with the food. If sinuses have formed, they

should be cleansed by injections of medicated solutions as advised for the mouth, and a mass of iodoform gauze and cotton should be placed over the openings and held in position by a bandage. Poultices and warm fomentations should not be applied to the face, as they increase the suppurative action and invite the flow of pus to the surface, resulting in the formation of sinuses and consequent disfigurement. Nothing should be done to interfere with the bone reproduction. No efforts at removal of the sequestrum should be made until the necrotic action is completed and the reproduced bone is sufficiently firm to preserve the shape of the original bone. In three of my earlier cases I endeavored to arrest the progress of the disease by cutting away the exposed process and scraping off the periosteum with the layers of new formed bone. In no instance was the operation successful in stopping the disease, and the deformity which followed the subsequent removal of the complete sequestrum was greater than in cases in which this plan of treatment was not attempted.

The sequestrum should be removed through the mouth, care being taken to separate the laminæ of new bone entirely, so

Fig. 14.

Sequestrum representing entire lower jaw. Right half removed ten weeks subsequent to removal of left half.

that they will not be fractured or drawn away with the sequestrum. When the disease has invaded the condyloid and coro-

noid processes of the lower jaw, the disarticulation is easily effected after incision of the structures in front of the ramus. In some instances it may be necessary to divide the ligaments of the articulation with the probe-pointed bistoury. When the entire lower jaw is involved, I have found it desirable in order to preserve the contour of the parts to remove but one-half at a time. After the expiration of eight or ten weeks the remaining portion may be removed. (Figs. 14, 15.)

Fig. 15.

Appearance of patient after removal of the entire lower jaw.

After the removal of the sequestrum the cavity between the two layers of new bone should be cleansed and packed with 7½ per cent. iodoform gauze; at each dressing the quantity used should be lessened in order to permit of the approximation and consolidation of the bone plates. For some time after removal of the sequestrum in the lower jaw a bandage should be applied so as to support the new bone. After removal of the sequestrum in the upper jaw the same form of dressing should be used until the cavity is closed by the fibrous tissue which forms, as a rule, in place of bone.

During the period of suppuration the strength of the patient is to be maintained by the administration of preparations of

iron and quinine, concentrated food and well-regulated exercise in the open air. Care should be taken to avoid exposure to cold, and the hygienic surroundings should be carefully attended to.

The prevention of the disease is to be accomplished by the joint efforts of employers and employés. The introduction into the manufactories of systems of ventilation which will promptly remove the phophorus fumes and as well the smoke from burning matches, will contribute materially to prevent the occurrence of the disease. While this may be accomplished successfully so far as relates to certain parts of the works, there are practical difficulties in the way of securing it in those rooms in which the matches are handled and constantly igniting, owing to the great danger which would arise from serious conflagrations if currents of air sufficient to remove the fumes and smoke were introduced. Efforts should, however, be made until some safe method is devised to accomplish this. In one factory with which I am familiar, exhaust fans have been arranged so as to remove the fumes promptly from over the dipping machines. Since the introduction of this plan the employés working at these machines have been free from disease. Experience has proven the value of turpentine in neutralizing the toxic effects of the phosphorus fumes, and, as in the factories in England and on the continent of Europe, the employés in this country should protect themselves by wearing open, wide-mouthed bottles containing this agent, suspended by straps around the neck so that they should rest upon the anterior surface of the chest, affording, in this way, ready inhalation of the vapor. At various points in these rooms, protected from fire, large shallow vessels containing turpentine should be placed in order that its vapor may come in contact with that of the phosphorus and neutralize it. The substitution of amorphous phosphorus which does not fume, for the common phosphorus in the manufacture of matches has been recommended. The chief objections to its use are the cost, which is stated to be double that of the common phosphorus, and its want of ready fusibility. The safety match which is being gradually introduced into use is stated to contain no phosphorus. The chemically charged paper attached to the boxes is said to contain

amorphous phosphorus, and the manufacture, therefore, of this form of match should be free from danger.

On the part of the employés immunity from danger can be secured by the use of turpentine, as suggested above, frequent inspection of the teeth, and cleanliness. Inspections of the teeth should be made frequently, all accumulations of tartar should be removed, and carious teeth carefully extracted or treated and filled; during the treatment of the teeth or immediately after extractions, the patient should not engage in work in the factory. Careful washing of the hands before taking food should be insisted upon and enforced by those in authority, and if it is necessary that the noon meal should be eaten in the factory, a special room should be provided for this purpose, separate from the work-rooms.

I feel quite confident from the investigations made by me in connection with the cases which have been under my care, that it is possible, by the adoption of proper methods of ventilation in the factories and the enforcement of proper regulations relating to the employment of antidotal agents and strict attention to cleanliness, to prevent the occurrence of the disease. It affords me pleasure to state that the largest manufacturing establishment in this country, The Diamond Match Company, which exercises the most liberal care of the operatives attacked with disease, is keenly alive to the importance of securing, by the adoption of all means available, the prevention of the disease.

I desire, in conclusion, to submit the following propositions:—

1. That the disease is a local expression of the constitutional condition produced by the inhalation of the vapor of phosphorus, and by particles of the agent taken into the system with the food by operatives in match factories who do not give proper attention to cleanliness of the hands.

2. That the introduction of the agent into the system is, as a rule, very gradual, and in such small quantities as to avoid the production of symptoms of acute poisoning; that, in this way, the chronic toxic condition of the system is induced, characterized chiefly by disintegration of the red-blood corpuscles and fatty degeneration of the arterial coats.

3. That the toxic condition precedes the jaw disease, as is shown by the fact that the disease does not attack operatives recently exposed to the action of the agent, but those who have been exposed for a period of years.

4. That examination of the teeth of operatives has shown that many who have caries, and have returned to work immediately after the extraction of teeth, have enjoyed immunity from the disease, showing that the agent had not attacked the periosteal tissue thus exposed. This was further shown by the fact that in one of the cases necrosis did not appear until three months after labor in the factory had ceased.

5. That individuals vary in their susceptibility to the action of the poison; for this reason many suffer immediately with acute symptoms, such as nausea, vomiting, etc., and are compelled to abandon work in the factories.

6. That the conditions under which experiments have been made on animals, to prove the absence of the disease until exposure of the periosteum and peri-alveolar tissue was effected, are not similar to those to which operatives in match factories are subjected.

7. That treatment of the disease in the primary stage is efficient and prevents its progress.

8. That the antidotal powers of turpentine have been established.

9. That the disease is to be prevented among operatives by the adoption of thorough methods of ventilation, stringent rules with regard to cleanliness, and the free disengagement of the vapor of turpentine in all the apartments of factories in which the fumes of phosphorus escape.

DISCUSSION.

Dr. Edward M. Moore, of Rochester, New York.

I have nothing to say in discussion of the paper, because I never saw a case of phosphorus-necrosis of the jaws, but I want to

ask a few questions, because the paper has excited my interest very deeply.

I notice that among the cases a good many of the subjects were in the epiphyseal age. In ordinary necrosis we almost invariably find that it stops at the epiphysis, and the articulation is preserved. Is that the case in this peculiar form of necrosis?

Another question; I have removed the whole jaw for necrosis in the epiphyseal age, after waiting long enough for the laminæ to be perfectly formed. After it was removed, there was no shrinking of the involucrum, except that which was necessary to bring the two surfaces together. Therefore the observation in reference to the contraction of the arch would not apply to cases where the new bone was sufficiently formed. How long has Dr. Mears been in the habit of waiting after the formation of this new bone?

One more question; after the joint is removed, what kind of a joint do you get? Is it a good joint?

The last question is this; it is well known that phosphorus has had the reputation of being an aphrodisiac. I should like to know whether there was anything of this kind in these cases.

Dr. Charles B. Nancrede, of Philadelphia, Pennsylvania.

I should like to make an important suggestion. There is no epiphysis in the lower jaw. It develops from two lateral halves. The development of the new joint is plain. This is a compound joint. The movement after removal of the jaw is of a hinge character, and takes place from the fibro-cartilage, and unless the intra-articular cartilage is removed we, of course, get a good joint.

Dr. Mears. As to the first question asked by Dr. Moore, I desire to state that of the specimens which I have of patients on whom I have operated, all of them with the exception of two have been removed from young persons. This bone, which I hold in my hand, was taken from a girl eighteen years of age, and, as far as this case is concerned, the necrosis has not been limited.

In regard to the period of time which it is proper to wait before operation, I usually wait about four months. I observe the case closely, however, and watch the development of new bone. When I am satisfied that the new bone formation is sufficiently strong to support the parts and retain the shape of the arch, then I interfere. I do not wait until the sequestrum becomes disintegrated; I think it

best to remove it as early as practicable, and thus prevent the development of hectic conditions. In regard to contraction of the arch, this is liable to take place where the entire bone is removed. I prefer in those cases where it is necessary to remove the entire jaw to remove one-half at one time, and at a later period remove the other half. In that way a firmer symphysis is obtained than when the entire bone is removed at one operation. I think that this is a very important point.

In regard to the restoration of the joint, I have found that in these cases the function of the joint seemed to be as good as before, and the jaw performed its duty satisfactorily. Patients do not complain of any interference with mastication or depression of the jaw. There is no pain.

As regards the aphrodisiac effects, I have investigated this as closely as I could by questioning the operatives, but as these are, to a great extent, females, it was difficult to obtain much information. I began my interrogations with the men; one was a married man, and had not observed that his work had any effect upon his sexual desires, if anything it seemed to interfere with virility. I have questioned the female operatives, and as far as I can learn they had experienced no change whatever. Two were married, and one had had two children, the other one child; both had been married five or six years. As regards the menstrual flow, I found that there was no more interference than would have been expected from the nature of the work, independent of the presence of phosphorus.

A DEVICE FOR ATMOSPHERIC PURIFICATION.

BY DAVID PRINCE, M.D.,
OF JACKSONVILLE, ILLINOIS.

It may be taken as proved by observation and experiment, that the reason why wounds once exposed to the air and afterwards closed, behave worse than if first made in a subcutaneous manner, is the exposure to the contact of some particulate material floating in the air.

The subcutaneous method of operating (when practicable) is a means of avoiding the contact of this material. That it is not gaseous, not oxygen, or carbonic acid gas, or any other gas, is proved on a small scale by the filtration of air through cotton and other substances, and its complete incapability of starting and continuing the processes of putrefaction after such treatment.

If it were possible to make a surgical operation in an atmosphere so filtered, it is probable that an open wound afterwards closed so as to secure cutaneous union by the first intention, would behave as well as if the wound had been made in the subcutaneous manner.

The irrigation of wounds by means of weak solutions of carbolic acid or of mercuric bichloride, is an attempt (generally successful) of annulling the activity of the floating particulate atmospheric material, but in the opening of serous cavities it is not entirely practicable to employ the douche in contact with all the exposed surfaces. In view of the incomplete applicability of the spray and of the douche in the attempt to avoid the evil effects of these atmospheric agents, it becomes a question whether or not the air can, on a sufficient scale, be deprived of these agents, so that an opening into the cavities of the body can be made and closed up again, under such circumstances

that the behavior will afterward be the same as would have resulted from subcutaneous incisions.

Taking a hint from observations recently made in Paris,[1] upon the effect of rainy weather upon the number of microbes floating in the atmosphere, it occurs to ask, whether or not it is practicable to subject the air entering an operating room to the influence of artificial showers in order to precipitate to the ground the whole or the greater part of these enemies of surgery.

They are known to be heavier than the air because they entirely disappear from the air within a tight box which has been for several months in one position. This principle of rest is of

[1] As to the number of microbes in the air, some reliable information is to be obtained from some recent observations by M. Miguel in a publication reviewed by M. Hache in the *Revue de Chirurgie* for May and June, 1884.

By these observations, what was known before in relation to the influence of the weather is further confirmed.

The spores of the mould and cryptogams are especially variable, being most prevalent in damp weather. On the other hand, the septic microbes are least abundant during the rainy periods and most abundant during the periods of drouth.

The average number of microbes found in the air at Mt. Souris (near Paris) was 84 to the cubic metre of air. In approaching the centre of the city, the number was found to increase, being ten times that outside of the city.

A series of observations was made at the top of the Pantheon, 250 feet high, which showed that the air was more pure there than at Mt. Souris, and ten times less charged with microbes than that on the surface in the centre of the city.

In the interior of the well-kept habitations, the microbes increase during cold weather at the same time that they diminish in the free atmosphere.

To give an example of the enormous accumulation in apparently well-kept houses in populous centres, observations were made in the fourth story of a house recently built, in a bedroom occupied only twelve hours of the twenty-four, and there was found to be the large number of 526 microbes to the cubic metre of air, while in a room similarly situated at Mt Souris there were only 325.

In hospitals, microbes have been found by M. Miguel to the number of 6300 to the cubic metre. At the Hôtel Dieu, 5123 in the summer and at the Hopital de la Pitie an average, by the year, of 11,100.

The microbes found in surgical wards abound especially in the form of micrococcus. The microbes cultivated and inoculated into animals generally proved harmless, but in some rabbits and guinea pigs, grave affections were produced.

The dust deposited upon objects was found to contain microbes to the number of 750,000 to 1,300,000 to the cubic metre.

no use to us, for the purification of the air of an operating room by this means is impracticable.

The dry filtration by means of cotton or other substances to entangle and arrest the particulate substances floating in the air, is not practicable on account of the rapidity necessary in the entrance and exit of the air in order to displace the agents entering from without during the progress of an operation, and those emanating from the occupants of the room. If, however, we can cause the air entering the room to pass though several showers of water, we have an expedient which may entangle these objects and carry them to the ground.

The following is a description of such a device :—

1. Basement.—On the right hand is an entrance ventilator, twenty inches in diameter, in which a steam jet is made to play in order to infiltrate the entering air with very fine globules of water. (2) (3)

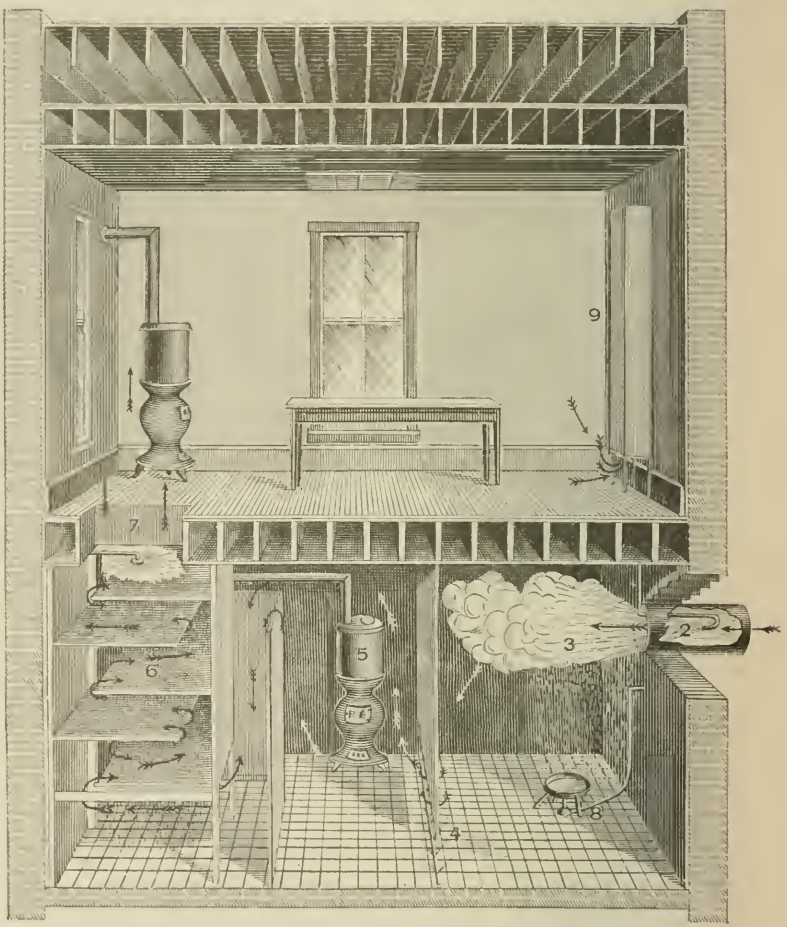
2. The air thus moistened passes in the direction of the arrows under a curtain or diaphragm reaching within fifteen inches of the floor. Under this curtain lies an iron pipe with numerous small holes drilled in the upper side, furnishing an interrupted spray under the curtain, and making it necessary for all the moving air to pass through this artificial shower near to the floor and into the next room warmed by the stove seen on the left hand of the diaphragm or screen. (5)

3. The air, thus warmed, passes through a filter, composed of shelves of thin muslin through which water is dripping. The warmed air first passes over the top of a vertical screen to avoid the entrance of cold air, then under another vertical screen, and then back and forth between horizontal screens fifteen inches apart, dripping with water, and finally through the spray which supplies this water. (6)

Thus there are three filtrations of the air, one by steam and two by water.

4. The air thus filtered three times, emerges through an opening in the floor and goes to the top of the operating room above. (7)

5. Operating Room.—The exit ventilation is seen on the opposite, or right hand side of the room, in the direction of the



Basement below and operating room above—scale, one to sixty.

(6) arrows. This is effected by a movable shaft or box, made by tacking muslin upon a frame, so that it can be set up against a window, with the upper sash pulled down to the necessary degree. The shaft can be shifted from one window to another to be free from the pressure of the wind, or on the opposite side, or from

the wind. The arrangement thus far is to get an atmosphere more pure than that outside, and through the frequent change of the air in the operating room to get rid, to the greatest possible degree, of the contamination of the air (during the progress of an operation) by the emanations from surgeons, assistants, spectators, and the patient himself. This change is secured by the entrance of filtered air from the basement, and the exit, from the floor of the operating room, of the air which has been the longest in the room, having descended gradually from the ceiling where the air is hottest, after having entered from the warming chamber below.

6. The floor of the operating room is made of yellow pine, and filled with paraffin to as great a depth as heated smoothing irons can drive it. By this means all cracks are filled so as to be non-absorbing. Under this lies a layer of tarred paper upon a common floor upon the joists. Between the joists lies a layer of tarred paper upon the ceiling, the under side of which ceiling is painted, and lined with muslin while the paint is fresh. The muslin is again painted on the under side. The floor thus has seven layers, including the joists.

7. The operating room is free from closets where anything unclean can be hidden, and all woodwork is either paraffined or painted.

8. There is no opening into any other room; about six feet of space intervening between the door of entrance and the nearest wall of the main building.

9. Before the use of the room for an operation involving the opening of a joint or the peritoneal cavity, it is intended that the rooms above and below shall be fumigated by sulphur burning in the basement. An iron pan for this purpose is figured in the cut (8) resting over a Bunsen burner for the combustion of sulphur. This is to be done for the destruction of any floating material of an organic character which may have gained entrance while the room may have been out of use.

It is found by trial that when the different sprays of water are going, one can remain very comfortably in the room above while sulphur is burning in the room below; the fumes being

first absorbed by the steam and having then to pass through the two different showers of water become thoroughly precipitated.

10. This building is the execution of a theory of combining the best known expedients for securing the best possible atmosphere for surgical operations, by excluding noxious agents and by destroying or expelling those which may have stolen in, or which may be introduced by the patients, or by the surgeon and his assistants and guests. It is supposed that enough air will enter and escape to change the whole volume of the air once in fifteen minutes. The exit draft coming from the floor will carry away most of the floating material.

11. The employment of a spray or douche of carbolic acid of the strength of 4 to 100, or of mercuric bichloride of 1 to 10,000, or other antiseptics, locally applied, though less necessary than in an ordinary room, may yet be resorted to in order not to omit any useful precaution.

12. The employment of solutions of carbolic acid, mercuric bichloride, or permanganate of potash, with the nail brush for cleaning the hands, should be supplemental to the fixed provisions against septic and pathogenic particulate infection.

13. The bathing of the instruments in carbolized water, while this proceeding is incapable of disinfecting or destroying any germs which may adhere to them, may yet be useful in an antiseptic sense, *i. e.*, by destroying microbes in a developed state or freeing them from the supposed secretion by which they may be surrounded, and which may serve as their weapon of attack by which they digest or destroy the surfaces with which they come directly in contact.

14. A useful precaution on the part of the operator may be a bath and a change of clothing, the hair and head being dampened so that dust will not escape from them; and yet another precaution may be the wearing of gowns which will oblige all dust escaping from the clothing of the operator and his assistants to fall to the floor whence it may escape with the draft which carries out the lowest stratum of the air.

NOTE.—The construction of an operating room on the principles here explained in a hospital already built, without erecting a detached building, would require

that two rooms should be taken one above the other, and if the upper one can have a skylight it will be of great advantage. The lower room might be in the basement with a shaft not less than (3) three feet in diameter extending to the upper room which might be under the roof for the advantage of a skylight. All communications must be closed by brick and mortar and an entrance opening made in the outer wall, the approach being secured by means of a platform on the outside of the building, so that it must be unavoidable to go into the open air for entrance and exit. The lower room should be equally shut off from communication with other rooms of the building.

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